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PREFACE TO THE EIGHTH EDITION.

The rapidity with which the seventh edition of this work became exhausted has necessitated the preparation of a new edition within a remarkably short space of time. Advantage has been taken of this condition of affairs to subject the text to a very careful reading, and revision where there is any possibility of obscurity in meaning or inaccuracy. A few new pages have been added. As the result, a new book is presented.

SAMUEL HORTON BROWN, M. D.

February, 1905.

PREFACE TO THE SEVENTH EDITION.

In the preparation of the seventh edition of this work, the previous edition has been subjected to most careful revision. The arrangement of the diseases has been changed considerably to conform with the more modern divisions of pathology. Introductory notes have been placed at the beginning of each section. Many new prescriptions and modern modes of therapy have been incorporated in the text. The method of expression has been largely reconstructed, except in the section on mental diseases, in which the colloquial style of the previous editions has, to a great extent, been retained. One hundred and thirty-seven pages have been added, and twenty-seven illustrations have been inserted. Obviously, the number of diseases considered is greater. A large index and an unusually comprehensive table of contents are contained within the book. The new material worthy of especial remark, includes the articles on the classification and general characteristics of fevers, the blood and its examination, examination of the sputum, examination of the stomach-contents, urinalysis, physical diagnosis, and the introductory notes on symptomatology placed at the beginning of each section. A table for the conversion of the apothecaries' system into the metric system has also been added. The typographical work is entirely new. In other words, the book has been completely revised and rewritten.

SAMUEL HORTON BROWN, M. D.

November, 1904. Philadelphia.

COMPEND

OF THE

PRACTICE OF MEDICINE.

INTRODUCTION.

The Practice of Medicine is the exercise of medical art, and embraces all that pertains to the knowledge, prevention, and cure of those departures from normal to which the term *disease* is applied.

Disease may be *organic* when there is structural change, or *functional* when there are no demonstrable lesions. It is questionable whether these forms can exist independently.

Pathology is the study of disease. It explains the origin and development (*pathogenesis*), causes (*etiology*), nature (*morbid anatomy*), and clinical history (*morbid physiology*) of the various abnormal conditions that may disturb the economy. It may be *general* when it is concerned with the study of morbid conditions common to a group of conditions or, *special* when it is restricted to the study of individual diseases.

Pathogenesis is that subdivision of pathology which treats of the origin and development of morbid processes or disease.

Etiology is that branch of general pathology which considers the causes of disease. These may be *internal, external, ordinary, specific, primary, secondary, predisposing,* and *exciting.*

The *internal* or *intrinsic causes,* include those having their origin in the mind such as prolonged mental application, intense or long-continued emotional excitement, long-continued mental depression, etc., and in the accumulation of certain products in the blood as the result of faulty secretion or excretion or the absorption of ptomains from the digestive tract.

The *external* or *extrinsic causes,* embrace traumatism and substances introduced into the body from without such as poisons, bacteria, toxins, etc.

The *ordinary causes* are those to which we are constantly exposed such as atmospheric and climatic changes.

The *specific* or *special causes* are in nearly every instance bacteria, each variety of which is capable of producing a distinct disease, for example, the *tubercle bacillus* producing tuberculosis, the *Comma bacillus* causing Asiatic cholera, and the *Plasmodium malariæ* giving rise to malaria. A disease produced by a specific cause or bacterium is said to be *infectious,* if the disease is communicable by contact it is spoken of as *contagious.* Infectious diseases may or may not be contagious but all contagious diseases are infectious.

A *primary cause* is the cause in which the affection took its origin. Traumatism is a common primary cause.

A *secondary cause* is a contributory cause and the term is usually applied to the various morbid excretory products of the blood.

The *predisposing causes* embrace any *inherited* or *acquired* susceptibility to disease.

An *inherited predisposition* is also called a *diathesis,* as examples of which may be mentioned the rheumatic and tubercular diatheses.

Acquired predisposition depends upon the race, sex, age, occupation, habits, and environment of the individual.

The *exciting causes* are those that immediately precede and precipitate an attack of any disease. The influence of atmospheric changes in the production of rheumatism may be mentioned as an example.

Morbid Anatomy or pathologic anatomy is that division of pathology which considers the structural change or *lesions* of disease. It may therefore be *gross* or *microscopic* (*histo-pathology*). Microscopic morbid anatomy may be said to include the study of the tissues (*histology*), the blood (*hematology*), and the various bacteria (*bacteriology*).

The Clinical History of a disease includes all the data referable to the manifestations of the disease process or its *morbid physiology*. It embraces the *symptomatology, physical signs, complications, sequels, diagnosis, prognosis, treatment,* and *termination*.

Symptomatology is the study of the various symptoms and signs whereby the disease is detected. They may be *objective*, when evident to the senses of the observer, such as redness, swelling, high temperature, etc., or *subjective* when the patient alone is aware of their existence, such as pain, numbness, vertigo, and nausea. The earliest recognizable symptoms are called the *prodromes*.

The Period of Incubation is the interval that exists between the entrance of a poison into the system and the manifestation of its symptoms.

Pathognomonic symptoms are those especially indicative of certain diseases as for instance, the rusty sputum of pneumonia and the eruption of small-pox.

An *acute disease* is one in which the invasion is sudden and rapid, and as a rule severe; when the symptoms develop less rapidly and are less intense, the disease is said to be *subacute;* when gradual or slow in development, of longer duration, and of lessened intensity, the disease is said to be *chronic*.

The physical signs are objective symptoms and are elicited by *inspection, mensuration, palpation, percussion,* and *auscultation*.

Complications are morbid conditions that may arise in the course of the original disease.

The sequels of a disease are the morbid phenomena which remain as the result of disease.

Diagnosis of disease, or the discrimination of diseases, implies a complete, exact, and comprehensive knowledge of the phenomena under consideration, as regards their origin, seat, extent, and nature.

A *direct diagnosis* is made when the morbid condition is revealed by a combination of clinical phenomena, or some one or more pathognomonic symptoms.

A *differential diagnosis* is the result when the diseases resembling each other are called to mind and eliminated from each other.

A *diagnosis by exclusion* is made by proving the absence of all diseases which might give rise to the symptoms observed, except one, the presence of which is not actually indicated by any positive symptoms.

The X-(or Röntgen) Rays, now in its infancy in diagnosis, will in the near future, doubtless, revolutionize the study of diseases of internal organs and structures.

The Prognosis of a disease is the conclusion or prediction relating to the future course or termination of the affection under consideration, or the art of making such predictions. Like diagnosis, it depends largely on clinical experience.

Treatment of disease may be *prophylactic* or preventive and *curative*. It may be divided into *hygienic, dietetic,* and *medicinal* treatment. It is *abortive* when the disease is checked in its early stage, *expectant* when the affection is allowed to pursue its natural course, *palliative* when the object is only to relieve suffering, and *restorative* when it aims to overcome weakness and prostration.

The termination of a disease may be in *cure*, *secondary processes*, or *death*. Cure may be affected by a slow return to health (*lysis*) or abruptly with a critical discharge (*crisis*).

Secondary processes are those in which the original affection is substituted by a new morbid process as in the case of endocarditis following rheumatism.

Death may be brought about by a progressively increasing debility (*asthenia*), as in phthisis, cancer, and Bright's disease; by an insufficient quantity or quality of the blood (*anemia*); by non-aeration of the blood (*apnea*) as in lung affections and croup; or by cerebral involvement (*coma*) as is seen in uremia, narcotic poisoning, and apoplexy.

Terminology.— Words ending in *itis* indicate inflammatory conditions such as *peritonitis*; those ending in *rhœa* or *rhea* refer to the transudation of liquid from a mucous surface, as for example *diarrhea*; those ending in *algia* denote painful conditions independent of inflammation as *gastralgia*; those ending in *æmia* or *emia* signify a morbid condition of the blood, as for instance *anemia*; those ending in *uria* relate to abnormal conditions of the urine as *albuminuria*; while those whose termination is in *oma* signify a tumor, for example *sarcoma* and *carcinoma*. A morbid condition of a part without any indication of its nature is designated by the suffix *pathy,* as *encephalopathy* and *adenopathy.*

The prefix *hydro* indicates a dropsical condition, as for instance *hydroperitoneum;* the prefix *pneumo* denotes the abnormal presence of air in a part, as *pneumothorax*; the prefix *peri* refers to the investing membrane of a part thus *perinephritis* indicates inflammation of the membrane surrounding the kidney. The connective tissue surrounding a part is designated by the prefix *para* as for example *parametritis,* the term for inflammation of the connective tissue surrounding the womb.

FEVERS.

Fever is a condition in which the body temperature is above normal (98.2°F.) and which is attended by quickened circulation and respiration, marked tissue change causing proportionate wasting of the body, and disordered secretions giving rise to anorexia, thirst, constipation, and scanty, high-colored urine of increased specific gravity. It may be due to a disorder of the sympathetic nervous system inducing disturbances of the vaso-motor filaments, or to a derangement of the nerve-centers adjacent to the corpus striatum which govern heat production, distribution, and dissemination. Fever may be said to result from a disturbance of the balance which normally exists between heat production and heat dissemination, and is usually toxemic in origin.

Rise of temperature is the most prominent feature of all fevers and can be accurately determined only by the use of the clinical thermometer placed in the mouth, axilla, rectum, or vagina. The mouth is usually selected by preference.

A fall of temperature below normal is a less frequent occurence but may be observed in collapse, cholera, convalescence from acute febrile diseases, and in chronic affections such as valvular heart-disease, myxedema, diabetes, certain nervous diseases, cancer, etc.

Degrees of Pyrexia:

Feverishness	99° to 100° F.
Slight Fever	100° to 101° F.
Moderate fever	101° to 103° F.
High fever	104° to 105° F.
Intense fever	105° to 106° F.
Hyperpyrexia	106° F. or over.

Fever may be divided into three stages: *invasion* in which the temperature gradually rises; *fastigium* or *stadium* in which its acme is reached and to some extent maintained; and *defervescence* or *decline* in which the temperature gradually drops until it becomes normal.

The decline of a fever may be by *lysis,* in which the temperature falls gradually or by *crisis,* in which it drops suddenly and is attended by *critical discharges,* as in erysipelas, malaria, measles, pneumonia, relapsing fever, and typhus fever.

Diurnal Variations (usually 1°F.) are common to all fevers. In most cases the highest point is reached in the early part of the evening (6 P. M.) and the lowest at a corresponding hour in the morning (6 A. M.), but occasionally this order is reversed.

Types.—Fever may be considered as of three types, *continued, remittent,* and *intermittent.* In *continued fever* the diurnal variation is seldom more than one or one and a half degrees. This type is seen in scarlet fever, pneumonia, and typhus fever.

In *remittent fever,* the diurnal variation is greater but the minimum temperature never reaches the normal point. This variety is observed in septic conditions, remittent fever, and typhoid fever.

In *intermittent fever,* the diurnal variation is very marked and the temperature drops to normal or below. As examples of this type may be mentioned the septic fevers, intermittent malaria, relapsing fever, and the fever associated with impacted gall-stones.

Some fevers are characterized by but one intermission or remission. For instance, variola has a remission on the third day, measles has a fall of temperature on the third or fourth day with a subsequent rise, dengue has an intermission on the third or fourth day which may extend over 48 or 72 hours, and yellow fever has an intermission on the third or fourth day.

The Pulse usually bears a direct relation to the temperature and in most cases a rise of one degree F. is attended by an increase of 8 to 10 beats of the pulse per minute.

A temperature of 98°F. corresponds to a pulse of 60
" " 99°F. " " " 70
" " 100°F. " " " 80
" " 101°F. " " " 90
" " 102°F. " " " 100
" " 103°F. " " " 110
" " 104°F. " " " 120
" " 105°F. " " " 130
" " 106°F. " " " 140

General Treatment of all Fevers.—All patients with fever should be placed at rest in bed in a moderately heated, quiet, and well-ventilated room. The patient should be nourished by the administration of milk, beef-tea, animal broths, and peptonized or other highly nutritious food in small quantities at frequent but regular intervals. Solids should be interdicted. The secretions should be rendered free by the administration of laxatives, diuretics, and diaphoretics.

The circulation should always be promptly brought within control. If the pulse is full, strong, and rapid, aconite or veratrum viride is indicated but if it is weak, drugs such as digitalis, caffein, and nitroglycerin are necessary. Alcohol is required only in long-continued fevers and those in which depression is marked.

The temperature may be reduced by hydrotherapy or drugs. Hydrotherapy includes the cold pack, the cold bath, and sponging. The drugs employed to reduce temperature are quinin, antipyrin, antifebrin, and phenacetin but their depressing action renders them somewhat dangerous, with the exception of the first named.

The Incubation Period varies greatly in the different fevers, as may be seen from the following table:

Asiatic Cholera	two to five days.
Diphtheria	two to six days.
Erysipelas	three to eight days.
German Measles	one to three weeks.
Measles	two weeks.
Mumps	two or three weeks or more.
Scarlet fever	four or five hours to a week.

Smallpox	ten days to two weeks.
Tetanus	24 or 48 hours to three weeks.
Typhoid fever	two to three weeks.
Typhus fever	four hours to two weeks.
Varicella	two weeks.
Yellow fever	two to three weeks.

Eruptive Fevers.—Certain fevers are attended by eruption, the date of the appearance of which is of extreme importance in the diagnosis.

The eruption of German Measles or Rötheln appears on the first or second day.
The eruption of Measles appears on the third or fourth day.
The eruption of Scarlet Fever appears on the first or second day.
The eruption of Smallpox appears on the third or fourth day.
The eruption of Typhoid Fever appears on the seventh or ninth day.
The eruption of Varicella appears on the first day.

Immunity.—Some of the infectious fevers confer protection against subsequent attacks among which may be mentioned German measles, measles, mumps, scarlet fever, small-pox, typhus, varicella, and yellow fever. Second attacks occasionally occur in measles, small-pox, typhoid, and typhus fever. The rest of the fevers seem devoid of immunizing properties.

Jaundice.—Ocasionally the disturbance of metabolism and tissue change are so great in fever as to interfere with the functional activity of the liver and a generalized yellowish discoloration or *jaundice* results. This is common in acute yellow atrophy of the liver, yellow fever, relapsing fever, and intermittent malaria.

SIMPLE CONTINUED FEVER.

Synonyms: Irritative fever; febricula; ephemeral fever; synocha.

Definition.—An acute, non-contagious, febrile disease of short duration and mild type unattended by characteristic lesions. It is most common in childhood and may arise from gastrointestinal disorders, mental or physical fatigue, excitement, emotion, or exposure to high degrees of heat or cold.

Symptoms.—The onset is sudden and may be ushered in with nausea, vomiting, convulsions, or a chill. It is attended by great lassitude. The temperature rises suddenly to 102° or 103°F. and is accompanied by headache, increased respiration, quick tense pulse, dryness of the skin, thirst, coated tongue, constipation, and scanty, high-colored urine of increased specific gravity. Delirium may be present in some cases. There is no constant or characteristic eruption but herpes are often observed on the lips. The duration of the affection varies from 24 hours to 6 or 7 days and may terminate by crisis or lysis. Convalescence is rapid.

Diagnosis.—The history is always of value in differentiating this condition from other somewhat similar affections, as most cases are observed in children as the result of mild gastrointestinal trouble. Local inflammatory conditions should be carefully excluded by a thorough examination. The concomitant symptoms will aid in distinguishing it from atypical cases of typhoid fever while the microscope will be of aid in differentiating it from remittent malarial fever.

Prognosis.—Uneventful recovery is the rule.

Treatment.—Rest in bed and a liquid or semisolid diet are essential. If due to gastrointestinal disturbances a powder containing calomel, gr. ⅙ (0.01 Gm.), sodium bicarbonate, gr. ii (0.13 Gm.), and powdered ipecac, gr. 1-12 (0.005 Gm.), should be taken every two hours until twelve have been consumed after which an enema or a seidlitz powder should be given. The body-surface should be sponged and diaphoretics and diuretics should be administered. Acetanilid gr. ii to v (0.13 to 0.3 Gm.), may be given every two or three hours in cases unassociated with digestive disorders. Aconite may be employed in drop doses when the pulse is quick. When the nervous symptoms and insomnia are marked potassium bromid, or trional may be used. During convalescence quinin and tincture of nux vomica are of great value.

INFLUENZA.

Synonyms.—La grippe; grip; contagious catarrh; epidemic or catarrhal fever.

Definition.—An acute, specific, infectious fever; moderately contagious; sporadic, epidemic, and pandemic; associated with catarrhal inflammation of the respiratory and sometimes of the digestive tract, muscular pain, disturbances of the nervous system and *debility* out of all proportion to the intensity of the fever and the catarrhal processes, and a tendency towards serious complications and sequels. There are no characteristic anatomic lesions.

The disease was almost unknown upon the appearance of the pandemic in the winter of 1889-90.

Causes.—The affection is induced by an extremely small, non-motile microörganism, the *bacillus of Pfeiffer* which is readily obtained from the sputum. The manner in which it produces the disease is not well understood. One attack seems to predispose to subsequent attacks. It usually occurs in epidemics along the lines of traffic.

Symptoms.—In the majority of cases the onset is sudden with a chill or chilliness followed by fever, the temperature reaching 101° to 103°F., a quick, compressible pulse, severe shooting pains in the eyes and forehead, and neuralgic pains in the joints and muscles. These symptoms are followed by chilliness along the spine, pain in the throat, hoarseness, deafness, coryza, sneezing, injected and watery eyes, and a dry irritative cough. The tongue is usually furred and anexoria, nausea, epigastric distress, vomiting, and sometimes diarrhea are present. Depression and debility disproportionate to the symptoms are almost constant. The symptoms usually group themselves so that an attack may be said to be of the catarrhal, gastrointestinal, or nervous type according to which group predominates. Any of these symptoms may be, and often are, greatly exaggerated causing the affection to simulate other febrile diseases. In mild cases the temperature falls on the fourth or fifth day by crisis and convalescence promptly begins in the absence of complications. Complications and relapses are common and frequently prolong the disease over several weeks.

Complications and Sequels.—Inflammatory conditions of the respiratory tract, especially pneumonia are the most frequent. Hyperpyrexia, cerebrospinal meningitis, pericarditis, and cardiac neuroses are also encountered as complications. As sequels may be mentioned phthisis, mania, confusional insanity, melancholia, neurasthenia, insomnia, neuritis, neuralgia, persistent headache, and lymphatic enlargements.

Diagnosis.—In order to recognize influenza, the sudden onset, marked general catarrh, the severe pains and pronounced prostration should be borne in mind. Isolated cases may be mistaken for acute bronchitis, typhoid fever, dengue, or cerebrospinal fever but the presence of these cardinal symptoms will serve to make the distinction.

Prognosis.—Recovery is the rule in young and healthy adults. According to Pepper the mortality is less than one half of one per cent in such cases. In either extreme of life the disease becomes proportionately more grave. The presence of chronic organic diseases such as Bright's disease, fatty heart, emphysema, and tuberculosis influence the affection unfavorably.

Treatment.—Supportive measures are indicated from the start to combat the marked exhaustion. The patient should be placed at absolute rest in bed and restricted to a semisolid diet. All the secretions should be disinfected. The bowel movements should be kept soluble, preferably by the administration of fractional doses of calomel. In the early stages a hot foot-bath or a hot-tub bath together with the administration of sweet spirits of niter or the solution of ammonium acetate may often be of great benefit.

The catarrhal symptoms and pains are often relieved by the following:

℞.	Phenacetin	gr. iii	.2 Gm.
	Pulv. camphoræ	gr. i	.065 Gm.
	Caffein. citrat.	gr. i	.065 Gm.

M. Disp. in capsul. vel chart. No. i.

S.—To be given every two hours alternated with quinin sulphate gr. ii (0.13).

Or—

℞.	Sodii benzoat.	ʒ ii	8. Gm.
	Salol	ʒ ss.	2. Gm.
	Phenacetin	gr. xl	2.6 Gm.
	Strych. sulph.	gr. ⅕	.012 Gm.

M. Disp. in chart. vel capsul. No. xii.

S.—One every three or four hours.

Antipyrin, salicin (R. G. Curtin), and quinin sulphate when administered during the very early stages may serve to abort the disease but should be carefully guarded to avoid intensifying the depression. In neuralgic cases the salicylate of cinchonidin in doses of gr. v (0.32 Gm.) every four hours is especially valuable.

Opium in some form may be necessary in severe cases to relieve the pains.

The frequent inhalation of the vapor from a pint of boiling water to which f℥ss (2 Cc.) of compound tincture of benzoin has been added, relieves the nasopharyngeal and bronchial symptoms but should they become troublesome the following mixture is advised:

℞.	Ammon. chlorid.........gr. x	.65 Gm.
	Tr. hyoscyam ....♏ xv	1. Cc.
	Syr. ipecac. ....♏ v	.3 Cc.
	Spts. frumenti.........fʒ ss	2. Cc.
	Aquae chloroformi .....fʒ iss	6. Cc.

M. S.—To be taken in water every three or four hours.

The complication of pneumonia is best combated by the use of stimulants such as alcohol and strychnin. The following formula is also recommended (Pepper):

℞.	Morphinæ sulph.........gr. j	.065 Gm.
	Quininæ sulph..........gr. xxxv	2.3 Gm.
	Strychninæ sulph........gr. ss	.03 Gm.
	Acid. phos. dil..........fʒ iij	12. Cc.
	Glycerini ....fʒ v	20. Cc.
	Aquæ ....q. s. ad f℥ iij	90. Cc.

M. S.—A teaspoonful four to six times daily, in water.

Bartholow advises the early use of pilocarpin, gr. 1/6 (0.01 Gm.), repeated until its mild physiologic effects ensue when it is substituted by duboisin, gr. 1-300 to 1-200 (0.00022 to 0.00032 Gm.), twice daily, and for the depression he employs the officinal pills of the iodid of iron, one pill every four hours and has the patient inhale one or two drops of pyridin every three or four hours.

During convalescence good food, and tonics such as strychnin, syrup of the iodid of iron, quinin, cod liver oil, etc., should be freely given with the view of preventing complications and sequels, which when they do occur, receive the same treatment as if they were independent affections.

TYPHOID FEVER.

Synonyms.—Enteric fever; gastric fever; nervous fever; enteromesenteric fever; abdominal typhus; autumnal fever.

Definition.—An acute, infectious, febrile affection, due to a special poison; characterized by insidious prodromes, epistaxis, dull headache followed by stupor and delirium, red tongue, becoming dry, brown, and cracked, abdominal tenderness, early diarrhea and tympany, and a peculiar eruption upon the abdomen; rapid prostration and slow convalescence; a constant lesion of Peyer's patches, the mesenteric glands, and the spleen with enlargement of the latter.

Causes.—The predisposing causes are early adult life (15 to 30 years), autumn season (late summer and early fall months), fatigue, and individual susceptibility (Pepper).

The exciting cause is the typhoid bacillus or *bacillus of Eberth*, which is found in the lesions, blood, stools, urine, and sputum of typhoid patients. The poison gains entrance to the system through the alimentary tract by means of contaminated water, milk, ice, meat, oysters, celery, lettuce, or similar substances. Carelessness in disposing of the stools is a frequent cause but flies may aid in the dissemination of the poison. The atmosphere is never impregnated with the fever germ.

Pathologic Anatomy.—The anatomic lesions of typhoid fever are invariably present and are characteristic. They consist in changes in Peyer's patches, solitary glands, and mesenteric glands and may be divided into three stages:

First. Stage of Infiltration or swelling due to excessive proliferation of the cellular elements and infiltration which also involves the surrounding mucous membrane. Peyer's patches become pale, thickened, hardened, and elevated above the mucous membrane. These changes may affect only three or four of the glands or may involve the entire number. They have been noted as early as the second day.

Second. Stage of Necrosis, softening, or sloughing of the diseased structures. The exudate may be absorbed or it may undergo necrotic changes and be discharged leaving an oval ulcer with an irregular margin having for its base the submucous, muscular, or peritoneal coat of the intestine. These changes take place in the second or third week of the disease.

Third. Stage of Ulceration, in which the separation of the sloughing and necrotic areas is complete leaving ulcers of various sizes at the sites of the Peyer's patches and solitary glands. This process belongs usually to the third week of the disease.

Fourth. Stage of Cicatrization, in which the ulcerated area is replaced by scar-tissue. The gland-structure is never regenerated.

In unfavorable cases, perforation is liable to occur at this stage. Under ordinary circumstances this stage is associated with the fourth week.

The mesenteric glands and spleen undergo changes similar to those in Peyer's patches, namely, infiltration, enlargement, and softening but seldom if ever rupture or ulcerate. The spleen usually begins to enlarge in the middle of the first week, the enlargement reaching its height at the end of the second week.

The mucous membrane of the entire intestinal tract is the seat of catarrhal changes and a similar condition is common in the respiratory tract. The heart, liver, and kidneys are affected with parenchymatous or granular changes.

In mild cases, the entire exudate in the lymphatic aggregations is absorbed without ulceration and in very rare instances the disease may manifest itself as a general septic infection without any anatomic lesions in the intestine.

Symptoms.—*Stage of Prodromes.*—The onset is insidious, with a feeling of general malaise, vertigo, headache, particularly occipital pain, disordered digestion, disturbed sleep, epistaxis, depression, and muscular weakness, followed by a chill or chilliness, the patient being unable to designate the day on which the symptoms began. In rare instances, the disease begins abruptly with a chill, followed by a high fever; this is particularly the case in malarial districts.

The exact duration of these premonitory symptoms is not known, and may be said to vary from a few days to two or more weeks.

First week, dates from the onset of the fever, when there are present increasing temperature, frequent pulse, headache, listlessness, the eyes closed as if asleep, coated tongue, nausea, diarrhea (there may be constipation), the abdomen moderately distended and, upon pressure in the right iliac fossa, gurgling sounds and tenderness. Upon the seventh day a few reddish spots resembling flea bites appear upon the abdomen, chest, or back.

Second week. The foregoing symptoms are exaggerated; fever is now continuous, with a frequent, compressible, dicrotic pulse, tympanitic, tender abdomen, gurgling in the right iliac fossa, nocturnal delirium, severe and constant headache, often stupor, a short

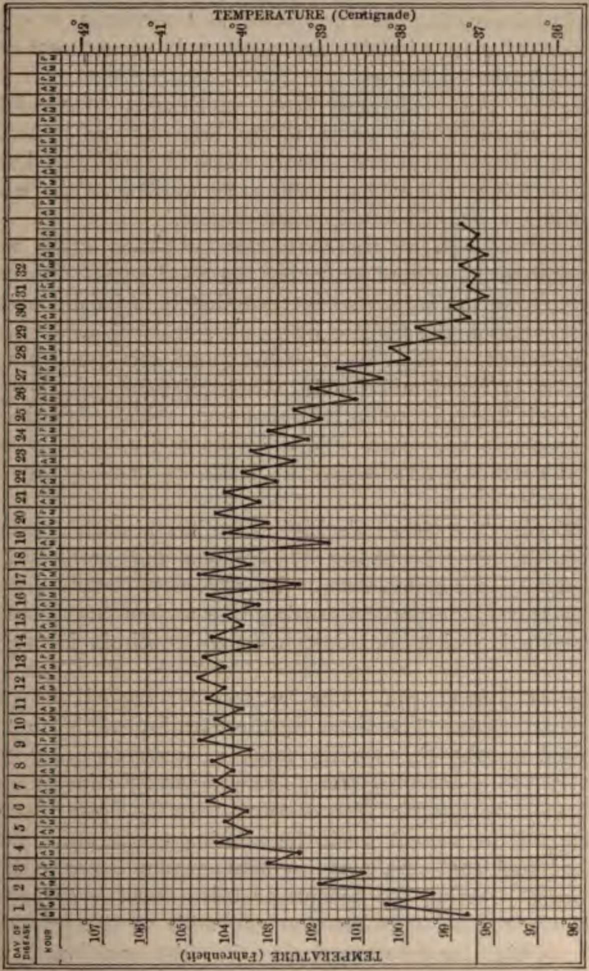

Fig. 1.— Clinical Chart of Enteric Fever of Four Weeks' Duration, without complications, which shows the temperature curve as uninfluenced by treatment. (From Wilcox's Fever Nursing.)

cough with distinct bronchial râles on auscultation, irregular muscular contractions (*subsultus tendinum*), sordes upon the teeth and lips, the tongue losing its coating and becoming more or less dry, the diarrhea continuing. During this stage deafness frequently develops, often increasing until profound, and persisting in convalescence. Disturbances of vision are common in pronounced cases. The spleen is increased in size.

Third Week. Fever changes from continuous to remittent; the evening exacerbations continue as high as the preceding week, the morning fall growing more decided each day, but all the other symptoms remain about the same until near the end of the week, when a marked amelioration begins.

In a fair proportion of cases all the symptoms grow worse toward the end of the second or during the third week. The prostration is extreme, the stupor so marked that it is hardly possible to rouse the patient, the tongue dry, hard, cracked, and covered with a brown crust, sordes collect on the gums, teeth, and cracked lips, the pulse is rapid and feeble, the respirations shallow and quickened, and there is retention of urine, which contains albumin. The stools are often voided involuntarily, and bed-sores develop, this condition terminating in death, or passing thus into the fourth week.

Fourth Week. The fever decidedly remits; almost normal in the morning, the pulse becoming less frequent and more full, the tongue gradually becoming clean, the abdomen lessens in size, the diarrhea ceases, the patient passing into a slow convalescence, greatly emaciated, which convalescence may continue for several weeks.

Analysis of Symptoms.—*The temperature record* of typhoid fever is characteristic. The fever on the morning of the first day may be stated at 98.5°F., evening 100.5°; second morning 99.5°, evening 101.5°; third morning 100.5°, evening 102.5°; fourth morning 101.5°, evening 103.5°; fifth evening 104.5°. From that time until the end of the second week the evening temperature ranges between 103° and 105°, the morning temperature being a degree or more lower. During the second or third week *hyperpyrexia*, or fever above 105°F., may develop and adds to the gravity of the attack. A high temperature during the third and fourth week is of grave import. Temperatures of 106°-107° with recovery have been reported, but are extremely rare.

Afebrile cases of typhoid fever are occasionally observed, all other symptoms including the prostration, excepting the step-like temperature, being present.

Diarrhea is the principal intestinal symptom; if absent, the lesion may be slight. The stools are at first dark, but early in the second week they become fluid, offensive, ochre-yellow in color, resembling "peasoup," and may be streaked with blood. They number from three to fifteen during the twenty-four hours.

Constipation occurs more frequently than is supposed. I have seen one hundred cases with constipation within the past ten years.

The *urine* has the ordinary febrile characters. Typhoid bacilli are demonstrable in about 20 per cent of cases. Retention is very common. Ehrlich describes a reaction which he believes is rarely met with save in typhoid fever. In examinations of the urine by Ehrlich's *diazo-reaction* in fifty cases of typhoid fever in the wards of the Philadelphia Hospital, the reaction was found in thirty-eight. It has also been found in a number of other conditions, particularly those having gastrointestinal symptoms. For its performance two solutions are necessary, one containing a saturated solution of sulphanilic acid in a solution of hydrochloric acid (50 Cc. to 1000), the other a 0.5 per cent solution of sodium nitrite. A few Cc. of the urine are then placed in a test tube with an equal quantity of a mixture of the first solution (40 Cc.) and the second solution (1 Cc.). The mixture is thoroughly shaken and covered with 1 Cc. of ammonia. A brownish red ring at the junction constitutes the diazo-reaction.

The eruption is almost constant. It consists of from five to twenty small rose-colored spots on the abdomen, chest, or back, sometimes on the limbs, appearing in crops, lasting about five days, disappearing on pressure and at death. It returns with relapses. Eruption day varies from the seventh to the ninth. Rarely spots of a delicate blue tint—the "*taches bleuâtres*" of French authors—are observed. Very occasionally in malignant cases it may become hemorrhagic or petechial in character.

Nervous symptoms are, pronounced headache, followed by dullness of intellect, passing into drowsiness and stupor, with great prostration. Deafness is pronounced. Sight is impaired, and in grave cases double vision results. Delirium, low and muttering, generally pleasant in character, is nearly always present in severe cases. *Coma vigil* is a grave symptom, the patient lying perfectly quiet with eyes open, taking no heed to his surroundings.

Splenic enlargement is an almost constant clinical feature. A vertical dullness exceeding two ribs and an interspace, signifies enlargement. Palpation is a valuable aid for determining splenic enlargement.

Muscular symptoms are developed late in the second or early in the third week, and consist of irregular contractions, carphologia, or *subsultus tendinum*, and are the result of the great debility. The reverse of muscular contractions, to wit, lying perfectly motionless in bed, attempting no muscular effort of any kind, is a grave sign.

Convalescence shows great debility and emaciation, extreme anemia, and severe nervousness, often very protracted. It is during convalescence that irritability of the heart, profuse night-sweats, insomnia, and in women loss of hair occur.

Complications.—*Intestinal hemorrhage* is the most frequent and at times the most critical of any of the complications of typhoid fever. The hemorrhage may occur any time between the fourteenth and twentieth day; a sudden decline of the temperature to the norm, or below, frequently precedes the passage of blood by stool. The hemorrhage is due to the erosion of a vessel during the ulcerative stage.

Perforation makes the case almost hopeless. It is attended by sudden localized pain and tenderness, tympanites, abrupt fall in the temperature, and symptoms of peritonitis (q. v.). *Peritonitis* without perforation adds to the gravity, but is not necessarily fatal.

Lobar pneumonia, hypostatic congestion, and *bronchitis* are frequent occurrences. There are few cases that do not have slight bronchial cough from the onset. *Albuminuria* and *acute nephritis* may occur, as may also *phlegmasia alba dolens,* the result of thrombosis of the femoral vein, usually the left. *Bed-sores* are frequent, resulting from the impaired nutrition, emaciation, pressure over bony prominences, and uncleanliness.

Ulceration of the tongue and mucous membrane of the cheek is sometimes observed.

Sequelæ.—*Paralysis,*—either mono- or paraplegia,—may take place due to an acute neuritis. *Post-febrile insanity* occurs more frequently after typhoid than any other febrile condition, except influenza. *Acute Nephritis* associated with edema, *alopecia,* complete or partial, *transverse markings of the nails,* and *tuberculosis* may develop.

Varieties.—*Abortive typhoid* is that variety in which convalescence is established within ten days or two weeks after an abrupt onset with marked symptoms. *Mild typhoid* is characterized by moderate fever, slight diarrhea, and few if any nervous symptoms. *Walking typhoid* is a mild type in which the symptoms are so slight as often to be disregarded by the patient. Cases of this character often terminate fatally from the very sudden occurrence of perforation and other serious complications. *Typhoid in children* is nearly always marked by the predominance of the nervous symptoms.

Relapses are not uncommon. The symptoms are nearly all repeated but are less intense than those of the original attack. A sudden elevation of temperature during convalescence independent of other symptoms is termed *recrudescence* and is due to excitement or gastrointestinal disturbances.

Diagnosis.—*The Widal reaction.* Widal and others have shown that "if to a drop of blood-serum, or to a drop of water containing a solution of dried blood from a typhoid patient, a moderate number of typhoid bacilli were added, a peculiar reaction occurred," seen under the microscope as a loss of the natural motility and an agglutination of the bacilli—a clumping into masses. The reaction seldom appears before the seventh or eighth day and may persist after recovery.

The characteristic symptoms that serve to distinguish typhoid fever from other diseases in which depression is a marked feature, are the temperature, the eruption, the diarrhea, and the enlarged spleen.

Enteritis has intestinal derangement and an irregular fever.

Peritonitis is attended with abdominal symptoms only, with constipation and rapid early prostration and collapse.

Acute miliary tuberculosis may be mistaken for typhoid fever. The temperature record is more irregular; there is no eruption;

the pulmonary symptoms are more pronounced; and the Widal and diazo-reactions are not obtained in tuberculosis.

Meningitis resembles typhoid fever somewhat but may be distinguished from it by its sudden onset, marked cerebral manifestations from the very beginning, leukocytosis, and the absence of the characteristic symptoms and reactions of typhoid already given.

Malaria may simulate typhoid fever but the blood examination will serve to clear up the diagnosis. Both diseases may be present coincidently in the same patient, but the mongrel affection *typhomalaria*, so-called, does not exist as such.

Concealed Suppuration will be distinguished by the fever chart and the leukocytosis.

Prognosis.—A positive prognosis cannot be made. Favorable indications are constipation, or slight diarrhea, low temperature, and moderate delirium. Unfavorable symptoms are obstinate and severe diarrhea, early high temperature, cardiac exhaustion, marked nervous symptoms with *coma vigil* or stupor, albuminuria, and repeated intestinal hemorrhages.

The prognosis is always more favorable in winter than in summer.

When death occurs, it is usually during or about the third week, the result of exhaustion, cardiac failure, or some complication. Children under puberty usually recover. More women than men die, although less women have the disease. Pregnant women and fleshy people usually succumb.

The mortality in typhoid fever in private practice is about one death in twenty; in hospital practice it varies from one death in five to ten cases, although the cold bath treatment has greatly reduced the hospital mortality.

Treatment.—The patient should be placed immediately in bed in a quiet, well-ventilated room having an average temperature of 65° F. Intelligent nursing is indispensable. The manner in which the disease is disseminated necessitates the most scrupulous cleanliness of the patient, the bedding, and the various sick-room requisites. The bed-pan should be employed through the entire course of the disease and the excreta may be rendered inocuous by adding twice their volume of chlorinated lime (one per cent solution) or carbolic acid (five per cent solution) and allowing the mixture to remain in a closed vessel for two or three hours before being finally disposed of through the sewer or by being buried. Bed-linen, or other clothing, that may have become contaminated should be disinfected by boiling.

The diet should be liquid and should be given in small quantities at intervals of two or three hours. Diluted milk, broths, soups, liquid peptonoids, coffee, tea, buttermilk, junket, albumin water, and similar foods are permissible but milk is undoubtedly the best. The appearance of curds in the stools indicates that the quantity of milk given is in excess. Usually it is best to dilute the milk with water, adding a small quantity of lime water, or to administer it mixed with some carbonated water. The average quantity of milk to be given at one time is about 5 ounces. Cool water may be given in small quantities to allay the thirst. Washing the tongue, lips, and mouth are also effective in this respect. Prostration is avoided to a great extent by regular feeding every two hours, but should the heart begin to weaken and the pulse become soft, whisky or brandy, in half-ounce doses every three hours, should be administered, preferably with the milk so as to aid in the digestive process. The periods of nourishment and stimulation should be the same if possible and should not be interfered with by sleep. The inclusion of solid foods in the dietary should not be considered until the temperature has remained normal for at least one week.

The reduction of temperature is perhaps the most important indication in the management of this disease. This is best accomplished by hydrotherapy. *Cold sponging* with water or alcohol and water is often of value in mild cases and to be effective the surface should be left very wet, being careful not to expose too great a portion of the body-surface at a time. *The cold pack* is of value in cases attended by rather high temperatures (104° to 105° F.) and is employed when for any reason the tub-bath is impracticable. The bed should be protected by a rubber cloth, and the patient, with his clothing removed, should be wrapped in a sheet wrung out of cold water. The surface should be rubbed briskly through the sheet, and from time to time cold water is freely sprinkled over the sheet. Friction must be continued during the pack, and ice-cloths or cap placed on the head. The duration of the cold pack is determined by the temperature and the reactive powers of the patient. Collapse may be avoided by the administration of whiskey, or brandy, or the hypodermic injection of strychnin before or after the pack according to the patient's condition.

The *cold bath,* after the method of Brand, or "tubbing," has proven most prompt and decided for reducing temperature. It consists in the systematic employment of general cold baths, with frictions, whenever the temperature reaches 102.2° F. As often as the temperature, taken every three hours in the mouth or rectum, is over 102.2°, the patient receives a bath lasting fifteen or twenty minutes. He wears a thin muslin garment or is wrapped in a sheet; he is given a stimulant and carefully lifted into the bath of 65° or 70°, some cold water being poured over his head and shoulders to lessen the shock; the head rests on an air pillow, the body submerged to the neck. *During the whole period of the bath the patient must be briskly rubbed.* The friction and affusion are of value in preventing chill and cyanosis. After the bath the wet linen is quickly removed and the patient placed in bed, wrapped in a dry sheet, and covered with a blanket. A stimulant is again given after the bath, and if any tendency to cyanosis or heart-failure, a hypodermic injection of strychnin. The temperature is taken after the patient is placed in bed and again in half to three-quarters of an hour, and if not then 102°, is not again taken for three hours. Not more than eight such baths should be given in twenty-four hours. The good effects of the bath are seen in a reduction of temperature, clearer intellect, and lessening stupor and muscular twitching. Sleep usually follows a bath, with a general stimulating effect upon the heart and the nervous system. There are no counter-indications to the cold bath except intestinal hemorrhage and perforation.

The various antipyretic drugs, such as antipyrin, acetanilid, phenacetin, etc., while successful in reducing temperature, should *never* be substituted for the bath treatment as they add to the already intense exhaustion. Quinin sulphate in doses of 15 to 20 grains (1 to 1.3 Gm.) twice daily, is of value in that it tends to lower the fever and at the same time is tonic and more or less stimulating.

Diarrhea should not be checked unless it exceeds three or four stools in twenty-four hours, when the following may be used—

℞.	Bismuth. subnit. ....gr. xx	1.3	Gm.
	Acid carbol. ....♏j	.06	Cc.
	Tinct. opii deodorat......♏xv	1.	Cc.
	Mucil. acaciæ ....ʒ j	4.	Cc.
	Aquæ ....fʒ iij	12.	Cc.

M. S.—Every three or four hours.

Or—

℞.	Cupri sulph.............gr. ⅙	.011	Gm.
	Extracti opii ....gr. ¼	.016	Gm.

M. S.—In pill, every four hours.

At the onset of a suspected case of typhoid fever, when there are present coated tongue, fetid breath, anorexia, chilliness followed by feverishness or fever, nervousness, costiveness or frequent tenesmic stools, and general soreness associated with mental unrest and headache, excellent results follow the use of the following combination:

℞.	Hydrargyri chlor. mit....gr. viij	.52	Gm.
	Sodii bicarbonatis ......gr. xv	1.	Gm.
	Pulv. ipecacuanhæ ......gr. ij	.13	Gm.
	Salol ....gr. xv	1.	Gm.

M.—Ft. chart. No. xv.

S.—One powder every three hours until decided bowel action.

Or—

℞.	Salol ....gr. iij	.2	Gm.
	Bismuth. salicyl., basic..gr. v	.3	Gm.

M. S.—In powder, after each stool.

Or—

℞.	Acid. sulph. aromat.....♏xv	1.	Cc.
	Tinct. opii deodorat......♏x	.6	Cc.

M. S.—In water, every three hours.

Constipation in the course of the disease is best relieved by enemas.

Tympanites may be relieved by the application of cold compresses, an ice-bag, or a turpentine stupe to the abdomen. In extreme cases, the introduction of a soft rubber catheter high up in the rectum will afford relief. If the tympany is associated with constipation, ten minims of oil of turpentine and fifteen minims of castor oil in emulsion, administered every three or four hours, will prove very beneficial. The quantity of food should be lessened in many cases as the distention is often due to fermentation of undigested food.

Headache when excessive, may be relieved by the application of cold to the head and mustard to the neck and by foot-baths. Morphin and atropin hypodermically may be required. Leeches are rarely necessary.

Delirium is in a large extent prevented by combating the general exhaustion. The use of stimulants and hydrotherapy control it in most cases, but camphor, musk, or morphin may be required.

Insomnia is sometimes a very troublesome symptom and necessitates the employment of trional, sodium bromid, or even morphin.

Cystitis may occur in typhoid fever and should be carefully guarded against by daily examination over the bladder and irrigation with sterile boric acid solution on the first signs of vesical irritation.

Intestinal hemorrhage indicates absolute rest and suspension of cold bathing. The foot of the bed should be slightly elevated, and an ice-bag placed over the right iliac region. Morphin, gr. ¼, should be given hypodermically at once. Fluid extract of ergot, fʒi (4 Cc.), Monsel's solution, ♏v to x (0.3 to 0.6 Gm.) or oil of turpentine, ♏x (0.6 Cc.) should be administered every two hours. The quantity of food should be reduced to the minimum and in some cases feeding should be suspended for twelve hours or more.

Perforation and *Peritonitis* require hot applications to the abdomen, bold stimulation, and morphin hypodermically. This is the most serious complication and demands consideration of surgical interference as soon as detected. The early operations are attended with the best results.

Bed-sores are prevented by scrupulous cleanliness as regards the patient and the bed, and by the avoidance of uneven pressure such as caused by crumbs, wrinkled sheets, etc. Bathing with alcohol and changing of the patient's position frequently, are of value.

Lobar pneumonia and *bronchial catarrh* call for dry cups and the use of the following mixture:

℞.	Ammonii chlorid	ʒ ij	8. Gm.
	Strychninæ sulph.	gr. ⅓	.02 Gm.
	Spts. chloroformi	fʒ j	4. Cc.
	Aq. lauro-cerasi..q. s. ad	f℥ iv	120. Cc.

M. S.—Dessertspoonful every two, three, or four hours, diluted.

In all cases the patient should be supported by the administration of strychnin sulphate, gr. 1-32 (0.002 Gm.) every four hours, and if the debility becomes extreme aromatic spirits of ammonia, fʒi (4 Cc.) or spirits of chloroform, ♏ii (0.12 to 0.3 Cc.) may be given every two hours in addition. If the tongue becomes dry, brown and fissured the following formula will be found useful:

℞.	Olei terebinthinæ	f℥ ss	15. Cc.
	Mucil. acaciæ	q. s.	q. s.
	Ol. sassafras	♏xv	1. Cc.
	Aq. chloroformi..q. s. ad	f℥ iv	120. Cc.

M. S.—One teaspoonful every two or three hours, diluted.

Convalescence should be carefully guarded. The return to solid food should be extremely slow. Exercise should be of the most mild character for several weeks. Quinin and belladonna, internally, will serve to control cardiac palpitation and excessive sweating during this period. Any tendency towards diarrhea may be checked by nitrate of silver, nux vomica, or strychnin. The malt liquors are of value in prolonged convalescence.

PARATYPHOID FEVER.

An infectious fever produced by a special bacillus, intermediate between the typhoid and colon form called the paratyphoid bacillus, and possessing clinical features similar to those of typhoid but of milder type. Diarrhea and termination of fever by crisis are more common than in typhoid. Intestinal ulceration is never present. Purulent arthritis and myositis may occur as complications in this disease, but are very infrequent in typhoid. The blood serum in this disease gives an agglutination reaction with fresh cultures of the paratyphoid bacillus, but not with typhoid cultures. The outlook is more favorable than in typhoid fever. Treatment is the same.

TYPHUS FEVER.

Synonyms.—Contagious fever; ship fever; jail fever; exanthematic typhus (German); petechial typhus; spotted or putrid fever.

Definition.—An acute, infectious, febrile, epidemic disease; highly contagious, and characterized by sudden invasion, profound depression of the vital powers, sickening odor, and a peculiar maculated and petechial eruption, favorable cases terminating by crisis about the fourteenth day. There are no characteristic lesions.

Cause.—A special infecting germ, the character of which is unknown, and which is influenced by filth and overcrowding. The disease is rarely seen in the United States except in seaports, where it is brought by emigrants.

Pathology.—There are no constant lesions peculiar to this affection. The blood is dark and thin with a decrease in fibrin and the tissues are affected with parenchymatous degenerations.

Symptoms.—After an incubation period varying from a few hours to two weeks the disease makes its appearance suddenly with a chill followed by pains in the head, back, and limbs, and fever, the temperature reaching 104° or 105° F. within a few days. The high temperature is maintained for about two weeks when it falls by crisis. The pulse is at first frequent and bounding but soon becomes small, weak and rapid. Prostration is extreme and is manifested by muscular feebleness, vertigo, tremor, and subsultus. On the fifth to the seventh day a coarse, red, diffused, measly eruption makes its appearance which rapidly becomes petechial or hemorrhagic. Associated with this is diffuse mottling of the skin which involves the entire body, excepting the face. The face has a uniform deep, dusky flush and the skin appears glazed. The conjunctivæ are injected and the pupils are contracted. As the disease progresses there is cutaneous hyperesthesia, muscular soreness, and tenderness over the tibia. Headache is severe and often followed by delirium. Constipation is the rule. The urine possesses no distinctive features.

Complications—Pneumonia, gangrene of the lungs, and swollen parotid glands are the most common.

Diagnosis.—It may be distinguished from *typhoid fever* by the history, the fever record, the course, appearance and distribution of the eruption, and the absence of abdominal symptoms, Widal reaction, and diazo-reaction.

Measles begins with coryza and cough and has an entirely different course except in the case of hemorrhage measles.

Cerebrospinal fever is attended by more intense nervous phenomena but there is no constant eruption. Prostration is not so great and vomiting is more common.

Prognosis.—The duration is usually about two weeks and the mortality varies from five to thirty-five percent. High temperature, frequent pulse, early stupor, and great anxiety are unfavorable indications.

Treatment.—Isolation is imperative. Disinfection of clothing and excreta is necessary. The patient should be treated in the open air if possible and the various symptoms combatted as in typhoid fever. Hydrotherapy should be employed to reduce the temperature but apart from this there is no special treatment.

CEREBROSPINAL FEVER.

Synonyms.—Epidemic cerebrospinal meningitis; epidemic cerebrospinal fever; spotted fever; cerebrospinal typhus.

Definition.— A malignant epidemic fever, characterized by headache, vomiting, painful contractions of the muscles of the back of the neck, retraction of the head, hyperesthesia, disorders of the special senses, delirium, stupor, coma, and frequently an eruption of petechiæ or purpuric spots. Lesions of cerebral and spinal membranes are found at the post-mortem.

Causes.—The disease is believed to be due to the *Diplococcus intracellularis* of Weichselbaum and also to mixed infection. The pneumococcus may also produce this disease. Among the predisposing causes may be mentioned bad hygiene, filth, overcrowding, foul air, poor food, impure water, exposure, winter season, and youth. It is but slightly contagious and the method of its transmission is unknown.

Pathologic Anatomy.—In nearly all cases there is hyperemia of the membranes of the brain and spinal cord followed by an exudation of lymph and an effusion of serum most marked at the base of the brain. The cranial and spinal nerves are similarly affected in severe cases. The lungs, spleen, stomach, liver, kidneys, bladder, and muscles are in various stages of congestion and parenchymatous degenerations. In some cases death results from profound toxemia before structural changes have taken place.

Symptoms.—*The common form* begins abruptly with a chill, excruciating headache, persistent nausea, vomiting, vertigo, and weakness. The muscles of the back of the neck soon become rigid and retracted. The muscles of the back are shortly involved in a similar manner, resulting in opisthotonus, or arching of the back. Kernig's sign (inability to straighten the leg completely when the thigh is flexed at a right angle with the pelvis, the patient being in the recumbent posture) is nearly always obtained. There is great restlessness and the surface of the body becomes hyperesthetic. Muscular cramps are common and convulsions and delirium are frequent. Arthritis is not uncommon. Involvement of the special nerves induces intolerance to light and sound, blindness, deafness, loss of senses of smell and taste, tremor of the eyeballs and paralysis of ocular muscles. The temperature and pulse record are irregular. Emaciation is usually present. A petechial or purpuric eruption makes its appearance from the first to the fifth day. Herpes facialis, erythema, or urticaria may also be present. The *tache cérébrale* is usually obtained. Leukocytosis is always present. The duration of this form is from a few hours to several weeks but usually it reaches its height in from three to eight days, passing into either stupor and coma, or into a protracted convalescence.

The fulminant form is characterized by chill, depression, and in a few hours collapse and death.

The abortive form consists of one or more pronounced characteristic symptoms during the course of an epidemic terminating in prompt recovery.

The chronic form is that in which the duration is unusually prolonged, attended by headache, gastric irritability, and vague pains commonly terminating in death from exhaustion.

Complications and Sequelæ.—The common complications are pneumonia, pleurisy, endocarditis, pericarditis, typhoid fever, polyarthritis, and intestinal catarrh. As sequels may be mentioned persistent headache, blindness, deafness, mental feebleness, chronic hydrocephalus, epilepsy, and various palsies.

Diagnosis.—*Typhoid fever* begins slowly and has a characteristic temperature, less headache and no muscular rigidity or opisthotonus. The eruption, diarrhea, absence of palsies and Kernig's sign, and the presence of Widal's reaction should serve to make the distinction.

Typhus fever has a definite course and eruption and is not attended by muscular rigidity, retraction, disorders of the special senses, or palsies.

Tubercular meningitis differs in that it is not epidemic, has no eruption, is preceded by long prodromes, runs a tedious course, and a primary focus of tuberculosis may usually be detected elsewhere in the body.

A careful history and examination will serve to differentiate it from *small-pox, influenza,* and *acute articular rheumatism* which it sometimes resembles.

Prognosis.—The mortality varies according to the epidemic from 20 to 75 percent. Severe cerebral symptoms are of unfavorable significance.

Treatment.—The treatment is symptomatic and supportive. Isolation is not absolutely necessary but the excreta, and the clothing should be disinfected. The patient should be placed in bed in sanitary quarters and nourished by milk, eggs, meat-juice, broths, etc., at regular intervals. Nutritive enemas may be necessary. Morphin sulphate, gr. ¼-½ (0.016-0.032 Gm.) should be given hypodermically every two hours or extract of opium, gr. i (0.065 Gm.), may be administered by the mouth every hour until the stage of effusion and its consequent pressure symptoms appear. Quinin sulphate and potassium iodid are then indicated. Da Costa alternates potassium bromid with opium, especially in children. The convulsions may be relieved by chloral, gr. xxx (2 Gm.) given as the occasion requires it. The coal-tar products are dangerous in this disease and should be used only with the greatest caution. Whiskey and brandy are indicated to combat collapse but should not be used in the early stage.

Locally, cold compresses and ice-bags should be applied to the head and spine and counterritation, cupping, and leeching over the spine may be employed. Repeated lumbar punctures are sometimes of value. Goldscheider advocates active movements of the patient while submerged in a bath at ordinary temperatures.

During convalescence tonics such as iron, quinin, strychnin, cod-liver oil, iodids, and electricity are indicated.

RELAPSING FEVER.

Synonyms.—Febris recurrens; famine fever; bilious typhoid fever; spirillum fever.

Definition.—An acute, infectious, contagious, epidemic, febrile disease, self-limited, characterized by a febrile paroxysm, lasting about six days, succeeded by an entire intermission of the same duration, which is in turn followed by a relapse similar to the first seizure.

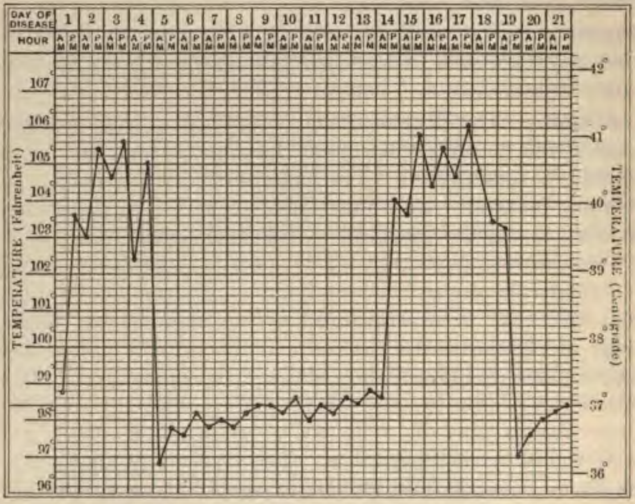

Fig. 2.—Clinical Chart of Relapsing Fever showing the febrile movement upon the tourteenth day. (From Wilcox's Fever Nursing.)

Cause.—The disease is due to the *Spirochæta Obermeieri*, a corkscrew-shaped microörganism. The predisposing factors in the production of this disease are overcrowding, bad hygiene, filth, poor food, impure air, and destitution.

Pathologic Anatomy.—There are no structural changes distinctive of this disease. The spleen is enlarged and usually covered with a fresh fibrinous exudation. The capsules are mottled and the splenic pulp is softened and shows enlarged Malpighian bodies. The liver and kidneys are swollen and congested. There may be catarrhal inflammation of the stomach and bile-ducts. The microorganisms are found in the blood only during the febrile paroxysms in the living subject. After death they may be found anywhere in the body.

Symptoms.—The onset is sudden with a chill followed by fever, 102°-104° F., frequent, rather weak pulse, headache, nausea, vomiting, and lancinating pains most marked in the calves of the legs. On the second day there is a sense of fullness in the upper part of the abdomen due to swelling of the liver and spleen. Jaundice is common. The fever falls by crisis on the seventh day to reappear on the fourteenth day, but with less severity. The symptoms then continue for about four days when convalescence slowly begins. There may be more than one relapse.

Complications.—Bronchitis, albuminuria, hematuria, paralysis, and ophthalmia are the more frequent complications.

Diagnosis.—The history, temperature record, and the presence of the microörganisms in the blood will serve to distinguish this affection from yellow fever, remittent fever, or any other disease with which it may be confounded.

Prognosis.—Recovery is the rule in uncomplicated cases.

Treatment.—Immediate isolation and disinfection are necessary to prevent the spreading of the disease. Rest in bed, liquid diet, and careful nursing are essential. The symptoms are treated, as they arise, on general principles, there being no special treatment.

MALARIA.

Definition.—An infectious fever, intermittent or remittent in type, characterized by enlargement of the spleen, chills, and anemia and due to the *Plasmodium malariæ* of Laveran.

Cause.—The exciting cause is the microörganism, already mentioned, which gains access to the body through the bites of mosquitos belonging to the genus *anopheles*. The predisposing causes are those factors that favor mosquito life, namely, marshy districts, high temperatures, humidity, and absence of winds. On account of the nocturnal habits of the *anopheles* the disease is more likely to be contracted at night.

Three forms of the microörganism have been recognized. The first or *tertian parasite* is characterized in the early period by small hyaline bodies possessed of ameboid movements. At first they occupy but a few of the red blood cells but as they increase in size and number they become surrounded by pigment granules. As the organism enlarges the pigment collects towards the center and the ameboid movements cease. Segmentation then begins and the parasite divides into from 15 to 25 parts or spores. The already distended blood cell now ruptures discharging the spores, into the blood stream, which repeat this cycle. The chills occur simultaneously with the discharge of the spores. For the completion of this cycle, 48 hours are usually required, so that a single group of these parasites induces a paroxysm every other day (*tertian fever*). The presence of two distinct groups sporulating on alternate days gives rise to a daily paroxysm (*quotidian fever*).

The second form or *quartan parasite* has less pigment of a more coarse quality, less spores, and its segmentation requires 72 hours. One group will cause a paroxysm every fourth day (*quartan fever*); two groups sporulating on two successive days, the paroxysms will occur on two successive days being separated by an interval of one day (*double quartan fever*). In the presence of three such groups, daily paroxysms will occur (*quotidian fever*).

The third or *estivo-autumnal parasite* is smaller, being about one-half the size of a red blood cell, and contains less pigment than the preceding. Within the blood cell it appears as a group of small hyaline bodies and soon causes the corpuscle containing it to assume a shrunken, crenated, and brassy appearance. After a week or more large ovoid bodies, crescentic in shape, appear in the corpuscles. Segmentation occurs only in the spleen and other internal organs. The entire cycle of this parasite covers 48 hours.

Flagellated organisms are sometimes observed during the evolution of the tertian and quartan parasites and are believed to be concerned in the reproductions of these organisms.

Pathologic Anatomy.—Disintegration of the blood cells is the most marked feature of the acute forms while in the chronic forms of permanent enlargement of the spleen from overgrowth of fibrous tissue is a common result.

Varieties.—The principal forms of malaria are intermittent fever, remittent fever, and pernicious malaria.

INTERMITTENT FEVER.

Intermittent fever is a variety of malaria, characterized by a cold, a hot, and a sweating stage, followed by an interval of complete intermission or apyrexia, varying in length according to the character and groups of the malarial organism.

Symptoms.—*The cold stage* begins with lassitude, yawning, headache, and nausea, followed by a severe chill in which the teeth chatter, the skin becomes pale, cold, and rough (*cutis anserina*), the nails and lips are blue, and the features are pinched. There is great thirst and the thermometer shows a rise of temperature to 102° to 104° F. These phenomena last from one-half hour to an hour.

The hot stage begins as the shivering ceases and the temperature rises still higher, 106° F. or more. The body-surface becomes hot and flushed and the pulse becomes rapid and full. Headache, backache, nausea, and intense thirst are also present. The urine is scanty, high-colored, and of increased specific gravity. This stage lasts from one to ten hours.

The sweating stage begins gradually, appearing first on the forehead and gradually extending over the entire surface of the body. All the symptoms subside as the perspiration becomes free. This period lasts from one to four hours and is often followed by a refreshing sleep.

An intermission, of varying length, then occurs after which another attack begins, being ushered in with chilliness or pain along one or more nerves.

Intermittent fever is attended by enlargement of the spleen, anemia, and pigmentation of the leukocytes but no increase in their number. It may be mistaken in a hasty examination for hectic fever, pyemia, or nervous chills but the finding of the organism in the blood will correct any error in diagnosis.

Prognosis.—Recovery is the rule with treatment. Neglected cases may terminate favorably after several paroxysms but are more likely to pass over into chronic malaria or malarial cachexia.

Treatment.—The cold stage may be, to a large extent, averted by the hypodermic injection of morphin sulphate, gr. ⅛-¼ (0.008-0.016 Gm.) or pilocarpin hydrochlorate, gr. ⅛ (0.008 Gm.), or by the internal administration of spirits of chloroform, fʒj (4 Cc.). During the hot stage cool drinks and cool sponging are indicated, and during the sweating stage the patient should be sponged with alum and hot water.

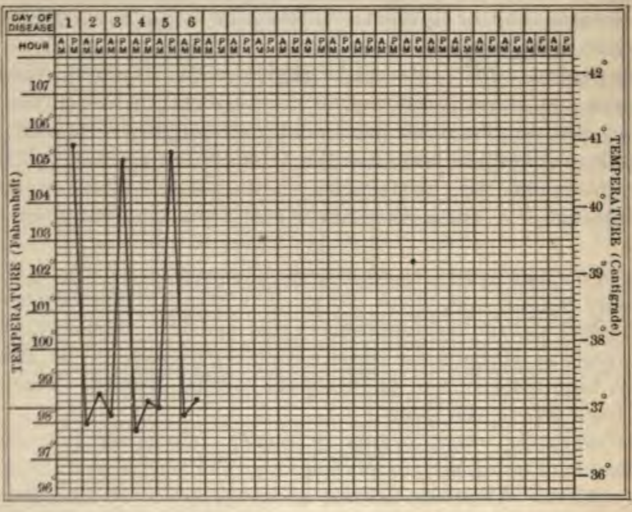

Fig. 3.—Clinical Chart of Ordinary or Tertian Malaria showing three febrile paroxysms occurring on alternate days. (From Wilcox's Fever Nursing.)

In the intermission the bowels should be opened by the administration of 5 grains (0.32 Gm.) each of calomel and sodium bicarbonate followed by an active saline cathartic. Quinin sulphate, gr. xvi-xxiv (1-1.6 Gm.), should be given in solution or pills in one or two doses, three to five hours before the expected paroxysm. Other preparations of cinchona may also be used.

After the paroxysms have been broken up, the solution of potassium arsenite (Fowler's solution), ♏v-x (0.3-0.6 Cc.), or the tincture of the chlorid of iron, ♏xx (1.3 Cc.) should be given every four hours over an extended period.

℞. Ferri reducti,

Quininæ sulph.	āā gr. lx	4. Gm.
Acidi arsenosi	gr. j	.065 Gm.
Ol. pip. nigr.	♏xv	1. Cc.

M. Ft. pil. No. xxx.

S.—One pill after meals, continued for one month or longer.

Relapses being common, it is well to administer quinin on the second or third day, fourth to the sixth, twelfth to the fourteenth, and nineteenth to the twenty-first day after the last paroxysm.

Preventive Measures.—The prevention of the disease has largely to do with exterminating mosquitos and avoiding infection of them and by them. The draining of stagnant pools and swamps with their subsequent filling up is well recognized as an effective measure. The use of crude petroleum over such surfaces has been found to destroy the larvas of the *anopheles,* in from two to four weeks and where practicable it should be employed. The screening of the patient and other individuals in malarial districts, during the sleeping hours, by means of ordinary netting is extremely efficacious. Sleeping on low ground, unprotected, should be avoided. Daily doses of from 5 to 10 grains of quinin sulphate is an additional protection.

REMITTENT FEVER.

Synonyms.—Bilious fever; bilious remittent fever; marsh fever; typho-malarial fever; estivo-autumnal fever.

Definition.—A paroxysmal fever, with exacerbations and remissions, in which the temperature is constantly above the normal; characterized by a moderate cold stage (which does not recur with each paroxysm); an intense hot stage, with violent headache and gastric irritability; and an almost imperceptible sweating stage, which is frequently wanting.

This variety of malaria lasts, as a rule, from seven to fourteen days, and usually occurs during the late summer and early autumn. Frequently the fever fails to remit and becomes continuous in type.

The characteristics that serve to distinguish this affection are the temperature record and the presence of the estivo-autumnal parasite already described. The spleen is always enlarged and there may be jaundice and delirium.

Prognosis.—Uncomplicated cases usually recover, but the disease may pass over into malarial cachexia or be followed by persistent headache and vertigo.

Treatment.—Quinin sulphate, gr. xvi (1 Gm.) per diem, should be administered by the mouth or rectum, or hypodermically during the remission. The following should also be given during this period:

℞. Hydrargyri chlor. mitis	gr. v	.3 Gm.
Sodii bicarb.	gr. v	.3 Gm.
Pulv. ipecac.	gr. ss	.03 Gm.

M. S.—To be taken as required according to the condition of the intestinal tract.

During the hot stage the patient should be sponged and an ice-bag placed on the head. If there is a tendency to cerebral congestion dry or wet cups should be applied to the nape of the neck and the following mixture given:

℞. Tinct. aconit. rad.	♏ij	.12 Cc.
Liq. ammon. acetat.	fʒ ij	8. Cc.
Liq. ammon. acetat.	fʒ ij	8. Cc.

M. S.—Every two hours.

The treatment advised in intermittent fever is also applicable to this variety of malaria.

PERNICIOUS MALARIAL FEVER.

Synonyms.—Congestive fever; malignant intermittent fever; malignant remittent fever; the congestive chill.

Definition.—A malignant, destructive malarial fever, which may be one of the intermittent or remittent form; characterized by intense congestion of one or more internal organs, together with dangerous perversion of the functions of innervation.

It occurs almost exclusively in warm climates and is due to the estivo-autumnal parasite. As a rule the pernicious character of the disease does not become manifest until after the second or third paroxysm.

Symptoms.—The disease begins as intermittent or remittent fever but with the migration of the parasites new groups of symptoms arise according to the localization of the organisms.

The gastroenteric type is characterized by intense nausea, vomiting, purging of thin discharge mixed with blood, tenesmus, burning sensations in the stomach, intense thirst, frequent weak pulse, cold hands, feet and face, shrunken features, cramps, and marked depression. It lasts from one-half to several hours.

The thoracic type is usually combined with the preceding and is attended by marked dyspnea, oppressed cough with blood-streaked sputa, frequent weak pulse, cold surface, and terror-stricken features, all of which arise from the intense pulmonary congestion.

The cerebral type is marked by violent delirium, followed by stupor and coma, slow full pulse, and a flushed or livid surface due to congestion of the brain.

The hemorrhagic type is caused by disintergration of the blood and is characterized by hemorrhages from the mucous membranes and into the subcutaneous tissues, bloody urine and jaundice.

The algid type is that in which the body-surface is intensely cold, the rectal temperature ranging from 104° to 107° F., a cold sweat covers the body, the pulse is slow, feeble, and often absent at the wrist, there is intense thirst, the mind is clear, and the countenance is death-like.

These various types exist usually more or less combined.

Diagnosis.—The predominence of certain of these groups of symptoms may cause the disease to be mistaken for cerebral apoplexy, meningitis, uremia, yellow fever, or cholera but a careful examination of the blood will reveal the characteristic parasite which is pathognomonic in malaria.

Prognosis.—The disease continues from a few hours to one, two, or three days and unless controlled prior to the second paroxysm is unfavorable. The intermittent forms are most favorable. The mortality is about 13 per cent from all forms of treatment.

Treatment.—The patient should be cinchonized immediately by the hypodermic injection of 40 grains (2.6 Gm.) of the bisulphate or dihydrochlorate of quinin. Bartholow advises the following:

℞. Quininæ disulph.	gr. l	3.3 Gm.
Acid sulph. dil.	♏ c	6.15 Cc.
Aquæ font.	f℥ j	30. Cc.
Acid. carbol. liq.	♏ v	.3 Cc.

M. S.—For hypodermic use.

The bimuriate carbamid of quinin is also highly recommended for hypodermic use.

The following formula, known as "Warburg's Tincture," has during the last few years gained considerable reputation in the various forms of malarial fevers:

℞. Rad. rhei, P. aloe soc., and Rad. angelica officinalis	āā℥ iv	120. Cc.
Rad. helenii, Crocus Hipsan., Sem. fœniculi, and Creta præparat.	āā℥ ij	60. Cc.
Rad. gentian, Rad. zedoar, P. cubeb, G. myrrh, G. camphor, and Boletus Laricis	āā℥ j	30. Cc.
Confect. damocratis*	℥ iv	120. Cc.
Quininæ sulph.	℥ lxxxij	2460. Gm.
Spts. vini rect.	Oxx	9600. Cc.
Aquæ puræ	Oxij	5760. Cc.

M.—Macerate in a water bath twelve hours, express, and filter.

S.—Warburg's Tincture.

Each half ounce (15 Cc.) contains quinin sulphate, gr. viiss (0.46 Gm.). If the stomach is too irritable to retain the tincture, the tincture may be evaporated to dryness and administered in capsules, each containing the equivalent of either one or two drachms.

In the cold stage, heat and stimulating lotions should be applied to the body-surface while in the hot stage, cold should be employed and morphin administered hypodermically.

In the gastroenteric type Da Costa recommends:

℞.	Morph. sulph.	gr. ¼	.016 Gm.
	Pulv. camph.	gr. j	.065 Gm.
	Mass. hydrarg.	gr. ij	.12 Gm.
	Pulv. capsici	gr. ss	.03 Gm.

M. S.—In pills every half hour until the character of the stool is changed.

For the thoracic type, dry or wet cups, carbonate of ammonia, caffein, and strychnin are indicated, while for the cerebral type venesection, cups or leeches to the neck, cold to the head, and prompt purgation, diuresis, and diaphoresis are required. For the algid type, morphin and atropin, hypodermically, ammonium carbonate and alcoholic stimulation are necessary but in the hemorrhagic variety, morphin, turpentine, dilute sulphuric acid, gallic acid, Monsel's solution, and the following are indicated:

---

*Formula of *Confectio damocratis*—

Cinnamon		14. Gm.
Myrrh		11. Gm.
White agaric, Spikenard, Ginger, Spanish saffron, Treacle, Mustard seed, Frankincense, and Chian Turpentine	āā	10. Gm.
Camel's hay, Costus arabacus, Zeodary, Indian leaf, Mace, French Lavender, Long pepper, Seeds of harwort, Juice of rape cistus, Strained storax, Opponex, Strained galbanum, Balsam of Gilead, Oil of nutmeg, Russian castor	āā	8. Gm.
Water germunder, Balsam tree fruit, Cubeb, White pepper, Seeds of carrot of Crete, Poley mont, Strained bdellium	āā	7. Gm.
Gentian root, Celtic hard, Leaves of Dittany of Crete, Red rose, Seeds of Macedonium, Parsley, Sweet-fennel seeds, Seeds of lesser cardamon, Gum arabic, Opium	āā	5. Gm.
Sweet flag, Wild valerian, Anise seed, Sagapernum	āā	3. Gm.
Spigrul, St. John's wort, Juice of acacia, Catechu, Dried bellies of skunk	āā	2.5 Gm.
Clarified honey		915. Gm.

The roots to be finely powdered and those whole mixed thoroughly.

℞.	Ext. ergotæ fld.........f℥ ss		15. Cc.
	Acid. sulph. dil.........fℨ iss		6. Cc.
	Acid. gallic ....ℨ j		4. Gm.
	Syr. zingib. ....fℨ iij		12. Cc.
	Aquæ .,.......q. s. ad f℥ iij	ad	90. Cc.

M. S.—Dessertspoonful every four hours, well diluted.

Malarial cachexia may result as a sequel to any of the forms of malaria just described. The patient is more or less jaundiced, the circulation is poor, the temperature is usually subnormal but there may be periodical attacks of fever, the spleen is enlarged, and weakness and emaciation are marked. Neuralgia, headache, hematuria, paraplegia, and orchitis may manifest themselves.

In the treatment of this condition iron, quinin, strychnin, arsenic, and cod-liver oil should be administered over an extended period. Occasional cinchonism is also necessary.

YELLOW FEVER.

Synonyms.—Yellow Jack; bilious malignant fever; typhus icterode; Mediterranean fever; sailor's fever; black vomit.

Definition.—An acute, infectious, paroxysmal disease, of three stages—the *febrile,* the *remission,* and the *collapse;* characterized by violent fever, yellowness of the surface, and "black or coffee-ground vomit."

Cause.—The disease is caused by a specific poison, in all probability the *Bacillus icteroides* of Sanerelli, the intermediate host of which is the mosquito—*Stegomyia fasciata.* No race, age, or sex is exempt from the disease. One attack confers immunity as a rule. It is essentially a tropical disease and is most common during June, July, August and September. The natives of warm countries, especially the negroes, enjoy comparative immunity to the disease but strangers are particularly susceptible.

Guitéras mentions three areas of infection: 1. The focal zone in which the disease is never absent, including Havana, Vera Cruz, Rio, and other Spanish-American ports. 2. Peri-focal zone or regions of periodic epidemics, including the ports of the tropical Atlantic in America and Africa. 3. The zone of accidental epidemics, between the parallels of 45° north and 35° south latitude.

Pathologic Anatomy.—Dissolution of the red blood cells and granular degeneration of the viscera are the most prominent structural changes. Jaundice, hemorrhages, and fatty degeneration follow these changes. A diagnosis cannot be made from the post-mortem lesions as none of them are distinctive.

Symptoms.—An incubation period of from 24 hours to 6 days precedes the attack.

The first stage or febrile stage is ushered in with malaise, headache, anorexia, or chill, followed by high fever, reaching shortly to 104° to 106° F., with pains in the head, limbs and back. The attack usually begins at night. The pulse is rapid, the face is flushed, the eyes are bright, and the stomach is irritable. As the temperature rises the pulse is at first more rapid but later shows a marked tendency to fall, a feature peculiarly characteristic. The patients are restless and anxious, and extremely prostrated. The urine is scanty, high-colored, acid, and contains albumin. Constipation is present. A peculiar and characteristic odor is emitted from the patient. The first stage lasts from thirty-six hours to three or four days, during the latter part of which the body becomes slightly icteroid. In severe attacks delirium is frequent.

The second stage is that in which the fever remits, the temperature declining to 100° to 99° F. All the distressing symptoms abate and the affection may terminate by crisis but more frequently after an interval varying from a few hours to one or four days it passes into the third stage.

The third stage, or that of secondary fever, is ushered in by a return of all the symptoms in an exaggerated form, followed by jaundice, which passes into a deep mahogany color, black vomit, hemorrhages from the mucous membranes, feeble pulse, cold surface, irregular respiration, and death from exhaustion, the mind remaining clear until the end. Recovery may occur even after the appearance of black vomit.

Diagnosis.—According to Guitéras the distinctive features of this disease are early jaundice and other *facies,* albuminuria, which shows itself even in mild cases, on the second, third, or fourth day, and slowing of the pulse as the temperature rises, most noticeable on the second or third day.

Dengue fever may be mistaken for yellow fever but it has but one paroxysm, the pains in the head, back, and limbs are more marked, and it lacks the early jaundice, albuminuria, and slow pulse.

Remittent fever may be distinguished by the presence of the malarial microörganism in the blood and the therapeutic test.

Acute yellow atrophy of the liver resembles yellow fever closely but the history, pulse, temperature, and presence of leucin and tyrosin in the urine will serve to differentiate the former from the latter.

Prognosis.—The disease seldom lasts more than one week. High fever, collapse, black vomit, and suppression of urine are unfavorable symptoms. The mortality ranges from 15 to 85 percent.

Treatment.—The spread of the disease should be prevented by screening the apartments of infected individuals and non-immunes by ordinary mosquito-netting. Swamps should be drained and covered with insecticides such as tobacco and petroleum, and the population of infected regions should be reduced to a minimum.

The indications are to keep the patient quiet in bed and to treat the symptoms as they arise. The irritability of the stomach permits of ingestion of food of only the most bland character. Mild laxatives such as castor oil, calomel, and citrate of magnesia may be employed in the early stages. Quinin should be given hypodermically. The gastric irritation may be relieved by cracked ice, carbolic acid, gr. ¼ (0.016 Gm.) in peppermint water, milk and lime-water, the application of a mustard plaster over the epigastrium, or—

℞.	Hydrargyri chlor. mitis..gr. 1-12	.005
	Morphinæ sulph. ....gr. 1-20	.003

M. S.—Every two hours until nausea is controlled.

The following prescription is employed by Surgeon-General Sternberg:

℞.	Sodii bicarb. ....gr. cl	10. Gm.
	Hydrargyri chloridi corr.gr. ⅓	.02 Gm.
	Aquæ destillat. ....Oij	950. Cc.

M. S.—Three tablespoonfuls every hour.

Stimulants such as alcohol, strychnin and digitalis should be administered to support the patient and the hemorrhagic tendency should be combatted by Monsel's solution, acetate of lead, adrenalin solution, and oil of turpentine. Enteroclysis is also advised.

The serum treatment has been employed by Sanarelli with questionable success. Out of 22 cases so treated, 6 died.

SCARLET FEVER.

Synonym.—Scarlatina, from the (old) Italian *scarlattina*, scarlatto (red).

Definition.—An acute, self-limited, contagious, infectious disease, characterized by high temperature, rapid pulse, a diffused scarlet eruption terminating with desquamation, inflammation of the mouth and throat, and frequently more or less grave nervous phenomena.

Cause.—It is due to a special microörganism as yet undetected but of exceedingly great vitality. It retains its infecting power for at least one year. The bearer of the contagion is in all probability the desquamated epithelium of the infected persons, the disease being communicable to the greatest degree during desquamation. The poison is disseminated by the scaly particles in the air, clothes or other fomites, food, etc. The respiratory tract is usually the route of infection but the digestive tract may also serve to carry the poison. Children are most likely to contract the disease. Second attacks are very uncommon but may occur.

Pathologic Anatomy.—The skin is the seat of acute inflammation which fades away in death. The liver, spleen, stomach, kidneys, heart, and muscles undergo granular changes. The throat is inflamed and ulceration sometimes occurs.

Symptoms.—The incubation period is short, varying from a few hours to a week, after which the affection manifests itself in one of three forms, *simple*, *anginoid*, and *malignant*.

The onset of the disease is sudden, being marked by a chill, vomiting, or convulsions, followed by pain in the throat, high fever, 105° F., and rapid pulse, 110 to 140 beats per minute. At the end of 24 hours *a bright scarlet rash* appears on the neck and chest, spreading over the entire body within a few hours. The eruption is not raised and disappears on pressure. Points of darker hue are scattered irregularly but there is no intervening healthy skin, the rash being uniformly distributed. The eruption may vary at times; occasionally it is scarcely visible; in some instances it may be slightly papular or vesicular (*scarlatina miliaris*); and in malignant cases it may be hemorrhagic or petechial. As soon as it is complete, it begins to fade, seldom lasting more than five days or a week, after which desquamation begins and occupies from two to six weeks. With the appearance of the rash the throat symptoms become prominent. Swallowing is difficult, there is pain and tenderness in the throat and jaws, the lymphatic glands are swollen, and inspection reveals a catarrhal inflammation of the pharynx and tonsils. A punctiform efflorescence on the tonsils, pillars, and pharyngeal vault may be observed before the rash appears. The tongue is at first furred, and later red with prominent papillæ—the "strawberry tongue." Headache, restlessness, and delirium may be present. Breathing is rapid. The appetite is lost and the bowels are usually constipated but diarrhea is not uncommon. The urine is scanty, high-colored, and often albuminous. Leukocytosis is present. The fever declines on the fourth or fifth day by lysis. The duration of simple uncomplicated cases is from three to fourteen days. Convalescence is slow.

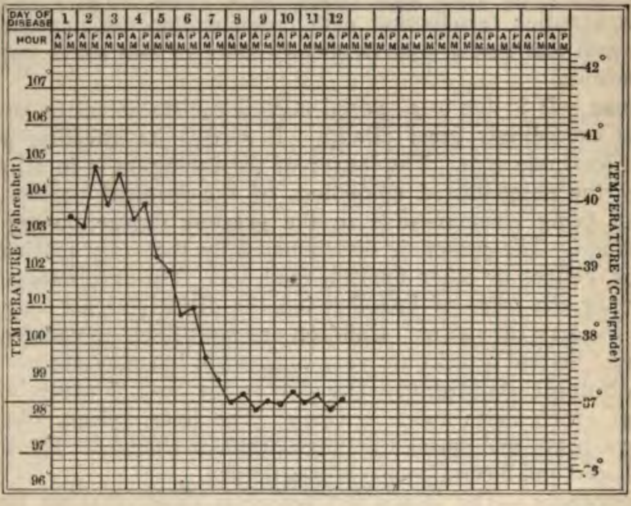

Fig. 4.—Clinical Chart of Scarlet Fever. (From Wilcox's Fever Nursing.)

Anginoid scarlet fever is marked by a predominance of the throat symptoms. There is high fever and great exhaustion. Frequently the enlarged glands suppurate.

Malignant scarlet fever is characterized by convulsions, delirium, muscular twitchings, high temperature, 107° to 110° F., rapid, feeble, and irregular pulse, and collapse. The eruption is of purplish color and in patches. Death may occur before its appearance.

Complications.—The principal complications are acute nephritis, arthritis, and otitis. Convalescence may be further complicated by chronic sore throat, diphtheria, ophthalmia, diarrhea, otorrhea, chorea, endocarditis, pericarditis, pleurisy, and suppuration of lymphatic glands.

Diagnosis.—The distinctive features of this disease are the high fever, rapid pulse, sore throat, and the early scarlet eruption with its subsequent scaly desquamation.

Measles differs from it in the character of its temperature, pulse, and eruption and is marked by a predominance of catarrhal symptoms.

Diphtheria resembles the anginoid variety but may be distinguished by the absence of the eruption and strawberry tongue and by the presence of the false membrane and the Klebs-Löffler bacillus.

Acute tonsilitis may be distinguished by the absence of the characteristic temperature, pulse, eruption, and tongue and the tendency towards nephritis.

Meningitis and malignant scarlet fever are somewhat similar but the history, mode of onset, pulse, and eruption will serve to make a diagnosis.

Erythema scarlatinoides has several points in common with scarlatina but may be distinguished from it by its non-contagious nature, the mild constitutional disturbance, the irregular distribution of the rash (the face being usually free), desquamation on the fourth day, the absence of the strawberry tongue, and the tendency towards recurrence.

Prognosis.—The mortality varies from 5 to 10 per cent. in mild epidemics to 20 to 30 percent in severe epidemics. The occurrence of complications adds to the gravity and uncertainty of the case.

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Treatment.—Isolation, rest in bed, liquid diet, and careful disinfection of all the sick-room articles is highly essential. The fever should be controlled by the cold bath (90° F.), douche, pack, or cool sponging. An ice-bag should be applied to the head. Drugs such as citrate of potassium, solution of ammonium acetate, spirit of nitrous ether, and tincture of aconite, alone or combined, may also be employed. The bowels should be kept regular by the administration of very small doses of calomel, soda, and ipecac, combined. Failing circulation will indicate the use of digitalis, strychnin, belladonna, alcohol, and the hot bath or pack.

With the appearance of the eruption the body should be anointed with cold cream, cocoa-butter, or the following:

℞.	Eucalyptol ....fʒ j		4. Cc.
	Petrolat. ....℥ j		32. Gm.

M. S.—Apply locally as directed.

In anginoid scarlet fever the following formula will be found valuable:

℞.	Tincturæ ferri chlor....fʒ ij		8. Cc.
	Glycerini ....f℥ j		30. Cc.
	Aquæ ....q. s. ad f℥ ij	ad	60. Cc.

M. S.—One-half to one teaspoonful every two hours, undiluted, according to the age.

Externally, in these cases, ice and cold compresses should be employed unless they produce discomfort, then heat should be substituted. Pellets of ice allowed to dissolve in the mouth often produce considerable relief. Dobell's solution should be used to spray the nasal fossæ and phraynx every hour.

℞.	Acid. carbolici ....fʒ iss		6. Cc.
	Sodii biboratis,		
	Sodii bicarb. ....āā ʒ ij		8. Gm.
	Glycerini ....f℥ ij		60. Cc.
	Aquæ ....q. s. ad Oij	ad	950. Cc.

M. S.—Dobell's Solution.

The following gargle may also be employed with benefit:

℞.	Thymol ....gr. iv		.26 Gm.
	Glycerini ....f℥ j		30. Cc.
	Aq. dest. ....f℥ j		30. Cc.

M. S.—A throat wash, dilute if necessary.

Peroxid of hydrogen, full strength or diluted, may also be used to antisepticize the mouth and throat.

In malignant scarlet fever, stimulation is the most important feature of the treatment. Whisky, brandy, iron, quinin, and strychnin should be administered to their physiologic limit.

Convulsions, restlessness, tremors, and other nervous phenomena are best controlled by hydrotherapeutic measures but the use of bromids, and chloral may be necessary in severe cases.

Serum treatment, using the antistreptococcic serum, has been employed by Moser, Charlton, and Hubbert with the view of preventing complications in this disease. Out of 15 severe cases, in which this treatment was employed these observers report but 2 deaths.

Acute nephritis is a common occurrence in scarlet fever particularly after desquamation, in the second, third, and fourth weeks. The urine should be examined daily in order to detect this complication as early as possible. Milk diet, digitalis, and protection of the patient from drafts are to a large extent preventive. With the development of nephritis the following formula should be employed:

℞. Potassii acetat. ....ʒ ij 8. Gm.
Spts. ætheris nitrosi....f℥ ss 15. Cc.
Aquæ ....q. s. ad f℥ ij ad 60. Cc.
M. S.—Teaspoonful every two hours, well diluted.

Or—

℞. Hydrargyri chlor. mitis,
Pulv. scillæ,
Pulv. digital. ....āā gr. 1/4-1/2 .016-.032 Gm.
M.—Ft. pil. No. j.
S.—One such pill every three or four hours.

Saline purgatives, dry cupping over the loins, warm baths, hot packs, vapor baths, enteroclysis, and pilocarpin will be indicated to relieve the system of the accumulated poisons. Convulsions will require the use of chloral, bromids, sodium benzoate, and chloroform in addition.

The scarlatinal arthritis will be benefitted by the alternate administration of iron and the following mixture:

℞. Ammonii salicyl.	ʒ ij	8. Gm.
Elix. simplicis	f℥ ss	15. Cc.
Syr. simplicis	f℥ j	30. Cc.
Tinct. card. comp.	f℥ ss.	15. Cc.

M. S.—Teaspoonful, diluted, four times daily.

In *otitis*, the application of a hot-water-bottle or syringing the canal with hot water will serve to lessen the pain but should the tympanic membrane bulge, it should be punctured to allow evacuation of any confined pus.

MEASLES.

Synonyms.—Morbilli; rubeola.

Definition.—An acute epidemic and contagious disease; characterized by catarrhal symptoms, referable to the naso-broncho-pulmonary mucous membrane, fever, and a crimson mottled, papular eruption which terminates by branny desquamation.

Cause.—The cause is an unknown microörganism apparently associated with the secretions of the respiratory tract. It is transmitted through clothes and other fomites. Children are especially predisposed to it but adults may be attacked. It usually occurs in epidemic but sporadic cases may be observed. Second attacks are very uncommon.

Pathologic Anatomy.—There are no characteristic structural changes. Catarrhal inflammation of the entire respiratory tract is almost a constant accompaniment. Gastrointestinal catarrh may also be present.

Symptoms.—After an incubation period of from ten to fourteen days the disease is manifested by a chill or chilliness, fever ranging from 101° to 102° F., muscular soreness, headache, and intense nasal, pharyngeal, and laryngeal catarrh. There are present also intolerance to light, redness and watering of the eyes, sneezing, and coughing. On the second day the fever remits, to rise again on the fourth day, when an eruption of small, dark-red, velvety papules arranged in crescentic groups, appears on the face and soon spreads over the entire body. The catarrhal symptoms still persist. The eruption is attended by itching and more or less burning and about the ninth day begins to fade and disappear entirely by bran-like desquamation. All the symptoms then gradually ameliorate.

Koplik has described a peculiar eruption consisting of small irregular spots of a bright red color, each having a bluish-white center, which appear on the mucous membrane of the lips and cheeks on the first day of invasion and which he believes to be pathognomonic. They fade away with the appearance of the dermal eruption.

Black measles, hemorrhagic measles, or *malignant measles* is that variety in which the eruption is hemorrhagic in character and there is profound prostration. It is encountered in camps, jails, and other places in which the hygiene is very poor.

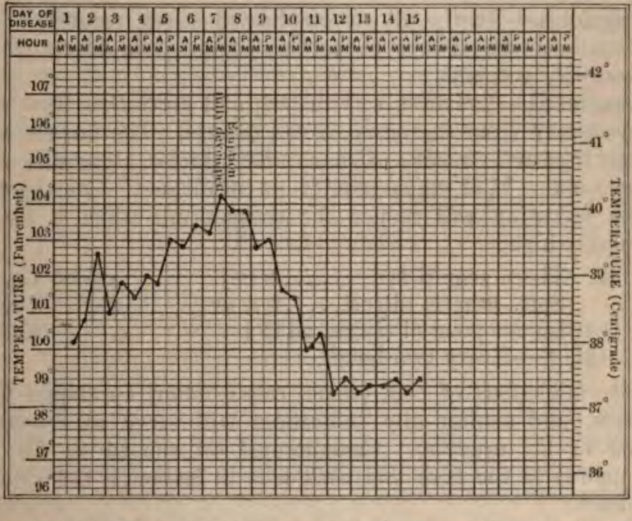

Fig. 5.—Clinical Chart of Measles showing defervescence by lysis beginning when the eruption is fully developed. (From Wilcox's Fever Nursing.)

Complications.—The most common complications are catarrhal pneumonia and gastroenteritis. As sequels may be mentioned tonsilitis, tuberculosis, ophthalmia, and cancrum oris.

Diagnosis.—The characteristic features of measles are its gradual onset, often with drowsiness, chilliness, nasal catarrh, watery eyes, fever which declines on the second day to rise on the fourth, the appearance of a crimson papular eruption on the fourth day preceded by Koplik's spots on the first day, and the bran-like desquamation.

Scarlet fever may be distinguished by the absence of Koplik's spots, and the difference in the character of the eruption, pulse, temperature, and symptoms.

German measles or rötheln may be diagnosed by the difference in the eruptions and the absence of constitutional manifestations.

Pityriasis rosea resembles measles somewhat but its rose-colored erythemato-squamous and papular patches are confined to the trunk and there are no constitutional disturbances as a rule. It is of longer duration.

Prognosis.—Nearly all uncomplicated cases recover. Lung complications are always of serious import. In black measles, the majority succumb.

Treatment.—Isolation, rest in bed, and protection from drafts are necessary from the onset of the disease. The bowels should be kept regular by means of some mild laxative. The diet should be semisolid. Mild cases require no medicines. Cool sponging or the following will reduce the temperature when it becomes alarmingly high:

℞.	Tinct. aconiti rad. ......♏ss-j	.03-.06 Cc.
	Spts. ætheris nitrosi.....♏x-xv	.6-1. Cc.
	Liquor potassii citrat., ad f℥ j	4. Cc.

M. S.—Every two hours.

Daily inunctions of cold cream, cocoa-butter, eucalyptol in petrolatum, and similar oily substances will serve to relieve the itching of the eruption. Camphorated oil rubbed on the chest and applied to the nose and neck aids in lessening the catarrhal symptoms.

During convalescence iron, strychnin, quinin, cod-liver oil, syrup of the iodid of iron, and similar tonics should be advised.

Black measles requires constant stimulation in addition to other measures. The various symptoms should be met with treatment as they arise.

RUBELLA.

Synonyms.—Rötheln; epidemic roseola; German measles; French measles; false measles.

Definition.—An acute, self-limited, contagious disease; characterized by mild fever, suffused eyes, cough, sore throat, enlargement of the lymphatic glands of the neck, and a rose-colored eruption, in patches of irregular size and shape, appearing on the first day.

Many so-called second attacks of measles and scarlet fever are attacks of rubella (Tyson).

Cause.—The disease is due to some special microörganism as yet undiscovered. It may be epidemic or sporadic. The contagion is disseminated by clothes and other fomites. Childhood is a predisposing factor. One attack usually confers immunity.

Symptoms.—The onset is sudden with mild fever, suffused eyes, little of no coryza, sore throat, enlargement of the cervical glands, and an eruption of rose-colored, pin-head sized spots which appear any time from the first to the fourth day. All the symptoms disappear within a week by lysis. Recovery is almost constant. There is no special treatment; the measures indicated under measles are applicable to this disease.

SMALL-POX.

Synonym.—Variola.

Definition.—An acute epidemic and contagious disease; characterized by severe lumbar pains, vomiting, and an initial fever, lasting from three to four days, followed by an eruption, at first papular, then vesicular, and afterward pustular; the development of the pustule being accompanied by a secondary fever, during the presence of which grave complications are prone to occur.

Causes.—A specific poison whose nature is unknown, maintaining its contagious vitality for a long period. There is no period, from the initial fever to the final desquamation, when the disease is not contagious, although the stage of suppuration is the most virulent. One attack, as a rule, protects from a second. Vaccination has a positive protective influence from the disease, an extensive observation having fully proven that in proportion to the efficiency of vaccination, is the rarity and mildness of variola.

Pathologic Anatomy.—A granular and fatty degeneration occurs in the liver, spleen, kidneys, and heart. The pustules are found in the larynx, trachea, bronchial tubes, and on the pleura.

Varieties.—Discrete; confluent; malignant; varioloid or modified small-pox.

Symptoms.—The manifestations of small-pox are preceded by an incubation period which varies from ten to sixteen days.

In the *discrete form,* the onset is sudden with a violent chill, vomiting, and agonizing pains in the back shooting down the limbs. The temperature rises reaching 103° to 104° F. within a short time.

The pulse is full, strong, and rapid ranging from 100 to 130. The face is red and the eyes are injected. Intense headache, sleeplessness, delirium, and convulsions may and often are present. Prostration is profound. On the third day the characteristic eruption appears first on the forehead and lips, consisting of coarse red spots. It may be preceded by a diffuse scarlatinous or measly rash (sometimes petechial in character) most marked on the inner surfaces of the arms and thighs. The true eruption becomes distinctly papular within 24 hours and the lesions acquire shot-like hardness. With the appearance of the eruption all the symptoms abate, the temperature falls several degrees, and the patient feels quite comfortable. On the sixth day of the disease the papules become converted into umbilicated and loculated vesicles, and on the eighth or ninth day these lose their umbilication and become mature pustules, each surrounded with a broad red band. The temperature again rises and the symptoms reappear at this period. There is marked edema of the skin between the lesions causing swelling of the surface and rendering the features unrecognizable. The tension of the pustules induces localized pains. On the tenth or eleventh day the pustules begin to dry up and are converted into scabs or crusts by the fourteenth day. These emit a peculiar offensive odor and fall off from the seventeenth to the twenty-first day leaving a red glistening depression or pit which soon changes into a white cicatrix. The secondary fever lasts about three of four days in favorable cases terminating by lysis.

Confluent small-pox is characterized by early appearance of the eruption, coalescence of the pustules, marked prostration, delirium, stupor, high and irregular secondary fever, swelling of the surface, and distortion of the features. Convalscence is tedious and disfiguring sequels are common in the most favorable cases.

Malignant small-pox consists in the appearance of widely distributed purpuric spots before the true eruption or in the occurrence of hemorrhages into the mature pustules. Bleeding from the mucous membranes is common. All the symptoms are intensified. This variety is also known as black small-pox and is nearly always fatal.

Varioloid is that modified variety of small-pox which occurs in vaccinated individuals or those who have previously been attacked. Its course is shorter and milder than other forms, the eruption appears later, and there is no secondary fever.

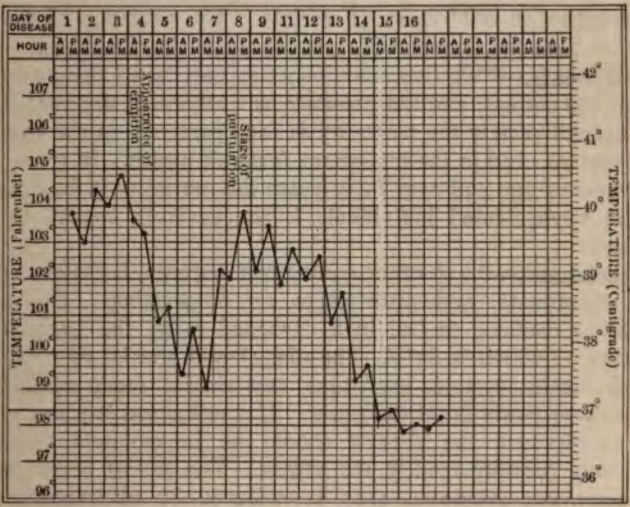

Fig. 6.—Clinical Chart of Small-pox showing fall of temperature upon the appearance of the eruption and its rise upon the incidence of the stage of pustulation. (From Wilcox's Fever Nursing.)

Complications.—During the course of the secondary fever there is a great tendency towards pleurisy, pneumonia, and dysentery. During convalscence boils, abscesses, ulcerative eye-diseases, otitis, and arthritis are prone to develop.

Diagnosis.—The characteristic features of this disease are the remittent type of fever, sudden onset with chill, vomiting, and excruciating pains in the back and legs, and the appearance of a papular eruption on the third day which later becomes vesicular and then pustular.

Varicella may resemble variola but the constitutional symptoms are less severe and the eruption which is essentially vesicular appears on the first day coming out in crops. The lesions dry up within two or three days.

Syphilis may be distinguished by its history, the polymorphous character of the eruption and its symmetric distribution, the adenopathy, the alopecia, and the mild constitutional symptoms.

Scabies is attended by papules and pustules and may simulate the eruption in mild cases of variola. The presence of the itch-mite between the fingers will serve to make the diagnosis.

Prognosis.—This depends upon the variety of the attack, the age of the patient, and the presence or absence of vaccination. In the discrete variety the mortality is four percent; in the confluent, fifty percent; and in the malignant form all perish. In those under five years of age and over forty years the mortality is fifty per cent and in unvaccinated individuals it ranges from twenty to sixty percent.

Treatment.—As in other contagious diseases, isolation, ventilation, cleanliness, and disinfection are imperative. The patient should be confined to bed in a darkened room, the average temperature of which is 65°F. The diet should consist of milk, eggs, animal broths, oysters, beef-juice, and similar foods administered every three hours from the onset. If vaccination has not already been performed, the patient should be immediately vaccinated as it may possibly modify the attack. The initial fever and the accompanying symptoms may be relieved by phenacetin, gr. x (0.65 Gm.), antifebrin, gr. v (0.32 Gm.), acetanilid, gr. v-x (0.32-0.65 Gm.), or antipyrin, gr. x (0.65 Gm.), repeated as the occasion requires it, avoiding depression. Headache may be controlled by the application of sinapisms to the neck and an ice-bag to the head. Sleeplessness, restlessness, and delirium indicate the employment of the bromids, trional, chloral, or opium. The irritability of the stomach may be overcome by ice pellets allowed to dissolve in the mouth, and the administration of dilute hydrocyanic acid, (♏ij). With the onset of the secondary fever, quinin, tincture of the chlorid of iron, and brandy should be administered in full doses. Hydrotherapy should be used to combat high temperature. The mouth and nasopharynx should be cleansed with Dobell's solution and boric acid lotion should be employed to irrigate the conjunctival sac.

To prevent pitting the patient should be kept in the dark and covered with some unctuous material or with cold wet dressings of bichlorid of mercury (1 : 5000 to 1 : 1000) or carbolic acid (10 gr. to the ounce). Hot water dressings may be more gratefully received. Painting of the pustules with ichthyol, five to twenty percent, is also recommended. Schamberg advises painting with iodin. Collodion is sometimes employed. When wet dressings are undesirable, eucalyptol in petrolatum (ʒi to ℥i) or carbolic acid and lanolin (gr. x to ʒi) may be applied.

Among the special forms of treatment may be mentioned the Finsen red-light treatment, the internal antiseptic treatment, the serum treatment, and antiseptic baths.

VACCINATION.

Synonyms.—Vaccinia; cow-pox.

Definition.—The reaction which follows inoculation with the vaccine virus or virus of cow-pox. It furnishes almost complete immunity against small-pox. It should be performed in infancy, at puberty, and whenever small-pox is prevalent.

Technic.—The area selected should be carefully cleansed with soap and water, and alcohol. The skin should be scratched and cross-scratched with an aseptic needle or special scarifyer being careful not to produce bleeding but instead merely oozing of pinkish serum. The virus is then rubbed in by means of the needle or scarifyer making additional scratches.

Symptoms.—Successful vaccination will be manifested on the third day by a papule, which becomes a vesicle on the sixth day, and a pustule on the eighth day surrounded by a reddish areola. The adjacent tissues are red and infiltrated. Tenderness and itching are also present. The areola begins to fade on the tenth day, and the pustule is converted into a mahogany-brown crust by the fourteenth day becoming detached about the twenty-third day. The resultant scar is circular, depressed, foveated, radiated and paler than the surrounding integument. In some cases, slight fever, malaise, restlessness, glandular enlargement, and other constitutional symptoms are present.

Complications.—Infection may occur resulting in abscess, erysipelas, or tetanus. Occasionally the eruption may be generalized. Sometimes it is followed by various eruptions resembling roseola, rubeola, urticaria, eczema, erythema multiforme, and similar affections.

VARICELLA.

Synonym.—Chicken-pox.

Definition.—A mild, slightly contagious, febrile affection; characterized by a moderate fever, and the appearance of a vesicular eruption which drys up and falls off in from three to five days. Its cause is unknown. Children are most often attacked.

Symptoms.—About two weeks usually elapses between the period of infection and the onset of the disease which is manifested by moderate fever, thirst, anorexia, and constipation. The eruption occurs within twenty-four hours at first being red spots which are rapidly converted into clear vesicles. The vesicles are not umbilicated or loculated and appear in crops. Itching is intense. The lesions are most abundant on the trunk; they dry rapidly dropping off within a week sometimes with pitting. Very rarely the vesicles become gangrenous.

The disease nearly always terminates favorably and without complications so that treatment is unnecessary except possibly to relieve aggravated symptoms.

ERYSIPELAS.

Synonyms.—Erysipelatous dermatitis; the rose; St. Anthony's fire.

Definition.—An acute, specific, infectious disease; characterized by more or less severe febrile reaction, and a peculiar inflammation of the skin generally of the neck and face. This inflammation exhibits a marked tendency to spread, to induce serious infiltration and suppuration of the areolar tissue, and to affect the lymphatic vessels and glands. Recurrences are common.

Cause.—The exciting cause is the *Streptococcus erysipelatis.* Lowered vitality, existence of abrasions and wounds, and the puerperal state are predisposing factors. It is feebly contagious and one attack predisposes to subsequent attacks. The incubation period varies from two to seven days.

Pathologic Anatomy.—The disease consists essentially of a septic inflammation of the skin and subcutaneous tissues, most frequently observed on the face and often directly traceable to some intranasal affection.

Symptoms.—The onset is sudden with chill, nausea, vomiting, or convulsions, malaise, headache, pains in the limbs, and a rise of temperature, 104° to 105° F. There is a corresponding increase in the pulse-rate. The tongue is coated and there may be diarrhea or constipation. The urine is scanty, albuminous, and high-colored. Delirium is frequent and in alcoholics resembles delirium tremens. Examination of the blood reveals a marked leukocytosis.

The eruption soon follows the intial chill and appears as red spots which rapidly coalesce forming a tense, crimson or violet-hued, shining area. This area is swollen and firm, is hot and tender to touch, and has a sharply defined border. Vesicles and blebs frequently develop. The patient complains of heat, tingling, burning, and itching in the affected tissues. The edema of the surrounding parts is marked, and when the face is involved distorts the features. The eruption begins to subside after five or six days followed by moderate desquamation and decline of the fever by lysis. When the eruption is attended by marked infiltration of the areolar tissues the term *phlegmonous erysipelas* is employed. When the affection is migratory in character, disappearing in one place to reappear in another it is called *erysipelas ambulans.*

Complications.—Complications are uncommon. Thrombosis of the cerebral vessels, edema of the larynx, septicemia, pneumonia, endocarditis, and rheumatism have been observed in the course of this disease.

Diagnosis.—The irregular fever, the early spreading eruption with burning, swelling, tension, and a sharply defined border, and the albuminous urine will distinguish erysipelas from the eruptive fevers, eczema, and erythema.

Prognosis.—The outlook is favorable except in alcoholics and in the migratory form of the disease.

Treatment.—Patients with erysipelas should be isolated from surgical and obstetrical cases on account of the contagious nature of the disease. In mild cases, the internal administration of a laxative and the tincture of the chlorid of iron with the local application of vaselin, ichthyol ointment (ʒi to ℥i), or bismuth oleate will suffice. In severe cases, the patient should be supported by the use of quinin sulphate, gr. ii (0.13 Gm.), extract of belladonna, gr. ¼ (0.016 Gm.), and tincture of the chlorid of iron, ♏x-xx (0.6-1.3 Cc.) every third hour. A liquid diet should be ordered. Alcohol may be required. Nervous symptoms should be combatted with appropriate measures as they arise.

Reynolds advocates in sthenic attacks the use of tincture of aconite, ♏ss-i (0.03-0.06 Cc.) every 15 minutes for the first two hours, then hourly until the surface is moist and the temperature lowered, being careful, meanwhile, to watch the condition of the kidneys.

Da Costa recommends pilocarpin hydrochlorate, gr. ⅙ (0.011 Gm.), hypodermically, or fluid extract of pilocarpin, ♏xx-xxx (1.3-2Cc.) every two hours in the rapidly spreading form of the disease. Iodid of potassium and the antistreptococcic serum have also yielded good results.

Local Treatment.—The following solution painted over the inflamed area and beyond, every two or three hours, sometimes seems beneficial:

℞. Acid. carbolic. ....f℥ ss		15. Cc.
Olei terebinthinæ ......f℥ j		30. Cc.
Tinct. iodi ....f℥ j		30. Cc.
Alcoholis ....f℥ j		30. Cc.
Glycerini ....f℥ ss		15. Cc.

M. S.—Apply locally.

Peroxid of hydrogen, glycerite of boroglycerin, lead-water and laudanum, carbolic acid lotion (ʒii to the pint), or silver nitrate solution (gr. xx to the ounce) may be applied with success. Ointments containing ichthyol, zinc oxid, mercurial ointment, eucalyptol, or soluble silver are also beneficial. The application of tincture of iodin or solid silver nitrate to the periphery often checks extension of the disease. In deep-seated varieties, scarifying and multiple incisions will be necesary.

DENGUE.

Synonyms.—Break-bone fever; neuralgic fever; dandy fever. The word *dengue* is pronounced *dangay.*

Definition.—An acute, epidemic, febrile disease, consisting of two paroxysms of fever with an intermission. The first paroxysm is characterized by high fever, distressing pains in the joints and muscles, and a peculiar eruption; the second paroxysm is characterized by a milder fever, an eruption of different character attended with intnse itching, by some recurrence of the joint pains, and by debility.

Cause.—Unknown; but it is evident that a peculiar condition of the atmosphere has some influence in its development. Incubation, from two to six days.

Symptoms.—Onset sudden—*fever,* 103° to 105°, intense headache, burning pains in the temples, backache, severe aching and swelling of the joints and stiffness of muscles, nausea, vomiting, constipation, and the appearance of a rash, resembling scarlatina. After some hours to two or three days a distinct intermission of one or two days' duration takes place.

The onset of the second paroxysm is also sudden, but the symptoms are much less severe, although the patient is greatly debilitated; it is at this time that the characteristic eruption appears, being either erythematous or rubeolous, and attended with intense itching, remaining for about two days, when desquamation occurs and convalscence is established, but is prolonged by the great debility of the patient. Average duration of the disease eight days. Relapses are common.

Diagnosis.—The history, course, paroxysmal character, and variability of the eruption will distinguish it from acute rheumatism, scarlet fever, and measles which it may sometimes resemble.

Treatment.—There is no special treatment. The disease nearly always pursues a favorable course. The symptoms should be treated on general principles. Laxatives, antipyretics, and analgesics are often indicated.

PAROTIDITIS.

Synonyms.—Parotitis; mumps.

Definition.—An acute, specific, infectious inflammation of one or both parotid and other salivary glands and the surrounding connective-tissue, with a very strong tendency to migrate into the mammæ or testes; characterized by pain, swelling, and disordered function of the glands. The affection is contagious.

Causes.—A specific poison. It occurs in epidemics, although isolated cases are seen. Males are more liable than females. The most common ages are between five years and puberty. As a rule it occurs but once in the same individual. The period of incubation is from two to three weeks.

Pathologic Anatomy.—There is inflammation of one or both parotid glands, and in severe epidemics the cellular tissue pervading the gland is involved. The catarrhal inflammation begins in the gland ducts and rapidly extends to the gland proper. There are congestion, swelling, and an infiltration of serous fluid, with more or less infiltration of the adjacent tissues. The swelling may suddenly reach an enormous size, and as suddenly decline, the gland returning to its normal condition, or, rarely, an abscess results, with partial or complete destruction of the gland. Occasionally the submaxillary gland, the ovaries, mammaæ, and testes are involved.

Metastatic parotiditis occurs secondary to severe blood-poisoning, as in pyemia, typhoid, or typhus fevers, or diphtheria. The usual termination of secondary parotiditis is by suppuration and destruction of gland-structure.

Symptoms.—The onset is rather sudden, attended by malaise, chill, fever, 101°-103°F., quick pulse, headache, dry skin, scanty urine, followed within a day or two by stiffness at the angles of the jaw, swelling of the parotid and other salivary glands, pain increased by moving the jaws, with general edema of the affected side of the face, at times the skin being reddened. Salivation is frequent, and occasionally deafness occurs. The swelling and other glandular symptoms subside about the sixth or seventh day, to be followed by restoration to health, or, what is more common, the involvement of the opposite gland.

At any time during the disease metastasis to the mammæ, ovaries, or testes is apt to occur, when the symptoms peculiar to such affections will be added. It has been noted that a continuance of the temperautre after the decline of the parotid symptoms has begun, usually is significant of metastasis. It is claimed that the involvement of other organs during the course of mumps is not an example of metastasis, but is a true transfer of the disease.

Prognosis.—In simple cases the prognosis is favorable. Metastasis to other organs may result in impairment of their functions.

Treatment.—Isolation with rest and liquid diet are the first indications. If the temperature is high, fever mixtures may be employed. Fluid extract of pilocarpin, ♏x-xxx (0.6-2 Cc.), may be administered. Locally, hot fomentations and ointments of belladonna, mercury, and guaiacol, alone or combined, are of value in relieving distress. If the condition tends to persist, blisters should be applied and potassium iodid administered. Orchitis will require the local application of ice, and mercury and belladonna ointments, and the internal use of tincture of pulsatilla, ♏iii-v (0.2-0.3 Cc.), every hour or potassium iodid.

DIPHTHERIA.

Synonyms.—Putrid sore throat; malignantulcerous sore throat; malignant quinsy; membranous angina.

Definition.—An acute, specific, constitutional disease; both epidemic and contagious, beginning by an affection of the throat, characterized by a local exudation and glandular enlargements; attended with fever, great prostration of the vital powers, and albuminuria, and often having for its sequelæ various paralyses.

Causes.—A specific germ, the Klebs-Loeffler bacillus. The bacillus in its growth produces a potent toxic substance—a toxalbumin, —the absorption of which produces the disease, and not the organism itself. The diptheria bacillus is associated with other pathogenic bacteria, the most active of which is the streptococcus pyogenes. It is preëminently a disease of childhood. It is apt to recur in those who have once been affected. All conditions of bad hygiene increase its virulence and diffusion, although the chief cause of its spread is contagion. Nasal, pharyngeal, and laryngeal catarrh produce a soil capable of promoting the growth of the bacillus and its toxin. The poison exists in the exudation and secretions of the fauces and saliva, and floats in the atmosphere at a considerable distance from the patient. The virus adheres to the clothing, the bedding, the furniture, and the room which the patient occupied. The period of incubation is from three to five days.

Pathologic Anatomy.—The diphtheritic inflammation differs from either the croupous or catarrhal form, in that the exudation is not only upon, but also within, the substance of the mucous membrane. At first there is redness, which may begin in any part of the throat, associated with swelling and an increased secretion of viscous mucus. The redness spreads over the entire mucous surface, when the exudation makes its appearance, at first giving the affected mucous membrane a glazed appearance, which is very characteristic. The deposit may commence from one or several points, such as one tonsil, the soft palate, or the back of the fauces, which, however, speedily extend and coalesce, forming extensive patches, or cover uniformly the entire surface. The patches are of variable thickness, which is increased by successive layers being formed underneath.

The color is usually gray, white, or slightly yellow, but may be brownish or blackish, the consistence ranging from "cream to wash leather." On removing the membrane, which is accomplished with more or less difficulty, a raw bleeding surface is exposed, and at times an ulcer, which is speedily covered with a fresh deposit. If the exudation separates itself, it is either not renewed at all or only in thinner films. The exudation or membrane, examined by the microscope, is composed of fibrin, pus corpuscles, epithelial granular cells, and the Klebs-Loeffler bacillus and other pathogenic bacteria. It is believed to be a product of coagulation necrosis.

If the larynx, trachea, or nasal mucous membranes participate in the disease, the croupous and not the diphtheritic form of inflammation occurs.

The lymphatic glands of the neck, whose vessels originate in the faucial tissues, are enlarged and inflamed, and contain large numbers of bacteria, probably originating as the result of decomposition.

The muscular tissue of the heart becomes soft, is easily torn, and its fibrillæ are far advanced in granular degeneration. Ulcerative endocarditis has been frequently observed. The kidneys undergo a granular degeneration in severe attacks. The blood undergoes alteration, becoming black and fluid.

Symptoms.—Following the law of contagious diseases, the symptoms vary in intensity in different cases, the prominent symptoms being often disproportionate to the gravity of the attack. The invasion may be mild, with rigors succeeded by moderate fever, headache, languor, loss of appetite, stiffness of the neck, tenderness about the angles of the jaw, or slight soreness of the throat. In other cases the invasion is more abrupt and severe, with chilliness followed by great febrile reaction, 103° to 105°F., pain in the ear, aching in the limbs, loss of strength, painful deglutition, and swelling of the neck, compelling the patient to take to bed from the onset. The appetite is poor, the tongue slightly coated, sometimes more or less exudation appearing upon it, the bowels either regular or slightly relaxed. The pulse at first full and strong, soon becomes either rapid or slow, but compressible. The urine is scanty, high-colored, and contains albumin. Prostration and weariness are present to a marked degree.

The *local symptoms* in the majority of cases are associated with the throat. The patient often complains of a frequent and persistent desire to hawk, in order to clear the throat. On inspection, the fauces are seen red and swollen and more or less covered with a film of diphtheritic exudation, giving a glazed appearance, soon followed by the dirty-white membrane; sometimes the tonsils and uvula are greatly swollen and spotted with exudation. Removal of the false membrane exposes to view a raw bleeding surface over which a new membrane promptly forms. In severe cases, more or less ulceration or sloughing may be observed. Not infrequently fragments of exudation, the false membrane, are expectorated, with particles of the ulcerated tissues, having an offensive odor, which is transmitted to the breath. The lymphatic glands of the neck are enlarged and tender, and in severe cases the tissues of the neck are greatly tumefied.

Extension to the nasal cavities causes a sanious and offensive discharge from the nose, with attacks of epistaxis. Constitutional reaction is marked. Enlargement of the deep faucial glands at the angle of the jaw is characteristic of this form of the disease.

Extension to the larynx is indicated by hoarseness or complete loss of voice, croupy cough, and obstructive dyspnea, which often becomes urgent, the breathing being noisy and stridulous, and subject to paroxysmal exacerbations. If the inflammation extend to the bronchi, the breathing becomes still more embarrassed. This variety runs a rapid course and often terminates in death by suffocation.

Duration.—The disease lasts from two to fourteen days, the average being about nine days, although complications and sequels may prolong its course. Relapses are not uncommon.

Complications and Sequelæ.—Eruptions on the skin, such as erythema, urticaria, and purpura may occur in the course of the disease and while not of serious importance may lead to errors in diagnosis. The most common complication is nephritis. Albuminuria is present in nearly all severe cases of diphtheria but when it is associated with blood-casts, epithelial casts, and scanty urine, the presence of parenchymatous nephritis is indicated. Capillary bronchitis, broncho-pneumonia, endocarditis, and arthritis also occur as complications.

After a severe attack patients often remain anemic and cachetic for an indefinite period. Paralysis, due to toxic neuritis, is a common sequel (10 to 20 percent) following the mild as well as the severe attacks. It may appear at the end of the first week but usually presents itself after convalescence has been established. It most frequently affects the phrayngeal muscles and palate seriously interfering with deglutition and impairing the voice. Anesthesia of the pharyngeal mucous membrane occurs coincidentally. The eye-muscles are affected next in frequency. Facial paralysis and palsy of the extremities may also occur. Sensation and reflexes are diminished in the paralysed parts. Neuritis of the cardiac nerves is not infrequent resulting in brachycardia, tachycardia, and even sudden cessation of the heart's action and death. In one case observed by the author the pulsations fell to 20 per minute. Multiple neuritis rarely occurs as a sequel.

Diagnosis.—The onset, course, throat symptoms, prostration, and the results of microscopic examination of cultures taken from the throat are the characteristics of this disease.

Intense *follicular tonsillitis* due to streptococcic infection may be mistaken for diphtheria. This exudate usually shows no tendency to spread and is in most cases limited to one tonsil, dropping out at the end of the second or third day leaving a clean cut ulcer which heals rapidly. Microscopic examination is diagnostic.

Scarlet fever may be confused with diphtheria but the characteristic eruption, strawberry tongue, rapid pulse, and the absence of the diphtheria bacillus will serve to distinguish them. They may exist coincidentally in the same patient.

From *membranous croup*, it may be distinguished by the difference in the constitutional symptoms; croup appears sporadically and is not contagious, diphtheria being highly contagious and frequently occurs in epidemics; in diphtheria of the larynx the depression is clearly that of blood-poisoning, while in croup the depression is in proportion to the mechanical obstruction of the respiration by the membranous exudation. The pathology of croup is simple and easy of investigation; diphtheria is obscure in its etiology and progress. The temperature record of croup is a high one until carbonic acid poisoning is imminent from the mechanical obstruction to respiration, while in diphtheria the tendency to a decline in the temperature after the fourth day is almost characteristic, regardless of the amount of laryngeal obstruction. In croup the pharynx contains no membrane, and is but slightly, if at all, inflamed, and associated trouble in the nose is of the rarest occurrence, the very reverse obtaining in diphtheria. In croup the laryngeal symptoms are present from the onset, while in laryngeal diphtheria the pharyngeal symptoms almost always precede. In croup glandular involvement is a clinical novelty, as are subsequent palsies, while glandular involvement and various palsies are the rule in diphtheria. The presence of the diphtheria bacillus in the membrane coughed up is conclusive evidence of diphtheria. Albuminuria is the rule in diphtheria, seldom occurring in croup.

Prognosis.—Always grave, but more so in children than in adults. Its gravity, in the majority of cases, is proportionate to the local symptoms. The average mortality is about ten percent.

Favorable indications are, moderate fever, strength slightly impaired, a good constitution, and moderate exudation.

Unfavorable indications are, high fever, great depression, spreading exudation, great swelling of the cervical glands, large amounts of albumin, extension to larynx and nasal mucous membranes, hemorrhages from the fauces and nose. and an epidemic character.

Treatment.—Antitoxin serum is indicated in all cases. It may also be employed as a prophylactic measure in those exposed to the contagion. The injections should be made where the skin is loose and at points that will not interfere with the patient's comfort. The dose is estimated in antitoxic units and not by the unit of the serum. The immunizing dose is from 300 to 500 units, the curative dose is from 1000 to 3000 units. If there is no improvement at the end of twenty-four hours a second dose should be administered. In addition to the antitoxin treatment supportive measures are indicated to combat the profound adynamia. The patient should be isolated and means taken to prevent spreading of the disease. Rest in bed with the employment of a diet composed of milk, eggs, broths, oysters, etc., every two or three hours is indispensable. If deglutition is painful or difficult, resort must be had to nutritive enemas, such as the following:

℞. Milk	f℥ j	30. Cc.
Spts. frumenti	fℨ iv	15. Cc.
Egg	One.	

M. S.—Add a small quantity of salt, mix thoroughly, and use as directed.

The room in which the patient is confined should be well-ventilated and its temperature maintained at an average of 70°F. The atmosphere should be rendered warm and moist by generating steam from an ordinary hot-water kettle, or by slaking lime in the sick-room. In the laryngeal form, direct inhalations of hot water vapor are indicated. Ice-pellets placed in the mouth afford great relief during these inhalations. Sponges dipped in hot water and applied to the angles of the jaw are also beneficial. To prevent dissemination of the poison by the exhaled air, Dr. J. Lewis Smith advises the following: Add four ounces of the following solution to one quart of water and allow them to simmer constantly, near the patient, in a broad-surfaced tin or zinc wash-basin: ℞. Olei eucalypt., acidi carbolici, āā f℥j (30 Cc.); spirit.terebinthinæ, f ℥ viij (240 Cc.). M. The vapor is strong, penetrating, and prophylactic, but not unpleasant. In hot weather, or when a fire is not convenient, saturate cloths a foot square with the same solution and place them on paper on the bed of the patient.

The medical treatment is general and local. Internally, stimulants should be used boldly from the onset; it is a mistake to wait for signs of debility before using alcohol in this disease. Other stimulants, such as strychnin, quinin, digitalis, nitroglycerin, and caffein should also be employed. Tincture of the chlorid of iron and bichlorid of mercury are used frequently in the following combination:

℞.	Hydrargyri chloridi corrosiv.	gr. 1-48	.0015 Gm.
	Tinct. ferri chloridi	♏ v-x	.3-.6 Cc.
	Glycerini	♏ x	.6 Cc.
	Aquæ	fʒ j	4. Cc.

M. S.—Every hour or two, well diluted.

The addition of tincture of belladonna, ♏ i-v (0.06-0.3 Cc.), to each dose increases its efficiency.

A combination of iron and potassium chlorate in full doses, frequently repeated, seems to modify the course of the malady and has the additional advantage of acting locally as it is swallowed. The following formula is frequently used:

℞.	Tinct. ferri chlor.	♏ v-x	.3-.6 Cc.
	Potassii chlor.	gr. iij-v	.2-.3 Gm.
	Glycerini	fʒ ss	2. Cc.
	Syr. zingib.	ad fʒ j-ij	ad 4.-8. Cc.

M. S.—In water every three hours, for a child of two or three years.

Calomel in small doses (gr. ⅙), combined with sodium bicarbonate every hour until spawn-like stools are produced, is beneficial, especially in cases showing a tendency to spread towards the larynx. Indeed, a tolerance to calomel seems to exist in laryngeal diphtheria.

In all cases the bowels should be kept regular by the use of laxatives, and the urine should be carefully watched throughout the entire course of the disease; diminution in the amount with considerable albumin is of grave significance.

Locally, measures should be employed tending towards the prevention of the spread of the infection. It is impossible to dissolve the false membrane in the throat by applications. Peroxid of hydrogen (50 percent), or Dobell's solution, used in a spray or on a cotton swab or sponge, should be freely employed every hour to keep the mouth and pharynx as aseptic as possible. Bichlorid of mercury (1 to 4000), carbolic acid (3 percent solution in equal parts of glycerin and water), salicylic acid (1 to 300), thymol (1 to 2000), lactic acid (30 gr. to the ounce), trypsin (30 gr. to the ounce), and papoid may also be used as local applications. The addition of tartaric acid to bichlorid of mercury increases its germicide properties. In a 1 to 500 solution the proportions are as follows: bichlorid of mercury, gr. 3.75 (0.25 Gm.), tartaric acid, gr. 19.25 (1.25 Gm.), water, 4 f℥ (120 Cc.). The following formulas are of value for local use:

℞.	Acidi carbolici	♏xx	1.3 Cc.
	Tinct. ferri chlor.	fʒ iv	15. Cc.
	Glycerini	f℥ j	30. Cc.
	Aq. destil.	f℥ j	30. Cc.

M. S.—Apply locally by means of a swab every three hours.

℞.	Potass. chlorat.	ʒ iv	15. Gm.
	Acid. carbol.	gr. ij-iv	.13-.26 Gm.
	Tinct. myrrh.	f℥ j	30. Cc.
	Inf. cinchonæ	f℥ ij	60. Cc.

M. S.—Use as a gargle or apply to throat with a cotton swab.

℞.	Menthol		10.0 Gm.
	Tuluol	q. s. ad	36.0 Cc.

M.—Et adde.

Ferri sesquioxid	4. Cc.
Alcohol absolut.	60. Cc.

M. S.—Löffler's Solution. Apply to throat by means of cotton swab.

Avoid struggling with children in an effort to forcibly spray or gargle the throat and nose; instead add glycerin to their internal medicine, and allow no liquids for some time after its administration.

In *laryngeal diphtheria,* the general treatment, especially the mercurial medication, should be the same. The patient should inhale the vapor of slaking lime and lime water (3 parts) and glycerin (one part) or liquor trypsin should be used as a spray every half hour or hour. When suffocation is threatened from the laryngeal obstruction, intubation or tracheotomy should be performed immediately.

Nasal diphtheria requires the same general treatment as the faucial variety, with additions of thorough cleansing of the nasal cavities every two hours with peroxid of hydrogen, carbolic acid, boric acid solution, Dobell's solution, potassium chlorate, or the following:

℞.	Sodii sulphit.	ʒ iij	12. Gm.
	Glycerini	fʒ ij	8. Cc.
	Aquæ	f℥ iv	120. Cc.

M. S.—Use locally as directed.

During convalescence in all forms, stimulation should be continued to prevent sudden heart-failure. Iron, quinin, strychnin, cod-liver oil, arsenic, etc., will be necessary to combat the attendant anemia and restore strength. Paralysis will necessitate massage and electricity in addition.

Prophylaxis.—As in other contagious diseases the patient should be isolated in a room stripped of all unnecessary furniture and draperies. Everything used by the patient or with which he comes in contact should be reserved for him alone. Instruments, tongue depressors, spoons, etc., should be boiled or kept immersed in carbolic acid solution. Bed linen, clothing, etc., should be sterilized by boiling or by exposure to superheated steam. Formaldehyde gas is employed for disinfection of the room (after the patient's removal) and its contents. All attendants should wear a gown of washable material on entering the sickroom, discarding the same on leaving it. The hands should be washed and immersed in an antiseptic solution before leaving the room. After convalescence is established, the patient should be washed with soap and hot water and then with alcohol (50 percent), carbolic acid solution (2 percent), or bichlorid of mercury (1 to 2000 solution) for three days in succession. The hair should be similarly treated or, in some cases cut off.

GLANDERS.

Synonyms.—Farcy; malleus humidus.

Definition.—An infectious disease of the horse, communicable to man and some domestic animals, but not to cattle; characterized by nodular growths in the nose—glanders, and under the skin—farcy.

Cause.—It is due to a specific bacillus—*Bacillus Mallei*. The organism resembles the tubercle bacillus, though somewhat shorter and thicker. The disease is communicated by the discharge from an infected animal to an abraded skin or mucous surface. Contagious. Incubation from three to five days.

Pathologic Anatomy.—Nodules, consisting of aggregations of round cells of lymphoid or polymorphonuclear type, which have a strong tendency to suppurative or necrotic softening. The floor and edges of the ulcers (softened nodules) are irregular and yellowish, discharging more or less purulent matter. The nodules develop particularly in the nares and the skin, and, rarely in the lungs. The lymphatic glands of the neck and elsewhere enlarge and may suppurate.

Symptoms.—There is an acute and a chronic form of glanders. *Acute glanders.* Redness and swelling of the nasal mucous membrane with burning and dryness, followed by the development of the nodules, which rapidly break down and discharge a fetid hemorrhagic or muco-pus. Soon there develop headache, painful deglutition, cough, fever, prostration, and typhoid symptoms, terminating eventually in death.

Acute farcy or glanders of the skin, consists of nodular swellings with subsequent ulcers and discharge of a fetid hemorrhagic pus on the skin. Papules, becoming pustules, followed by ulceration, occur in the neighborhood of the nodules. The lymphatic glands and vessels are involved, but the nose is not affected. Prostration and typhoid symptoms rapidly develop.

In the *chronic variety,* the development, course, and symptoms are all of less severity.

Diagnosis.—The certainty of diagnosis is made possible only by making cultures.

Prognosis.—Acute variety fatal. Chronic variety, if early diagnosed, many may recover.

Treatment.—Palliative and surgical means for the lesions. It is possible that the serum-therapy may soon be able to control, cure, or modify this fatal disease.

CHOLERA.

Synonyms.—Epidemic cholera; Asiatic cholera; malignant cholera; spasmodic cholera.

Definition.—An acute, specific, infectious disease occurring usually in epidemics, but may be endemic in certain localities, as in parts of India, characterized by violent vomiting and purging of a peculiar, rice-water-like fluid, severe muscular cramps, and a condition of prostration followed by collapse and death, or of reaction from collapse with the subsequent development of a typhoid state (cholera typhoid).

Causes.—A specific poison, the "comma bacillus" of Koch, which is found in great numbers in the discharges. Cholera is but feebly contagious, in the usual acceptation of that word, but it is unquestionably infectious.

The evidence seems conclusive that the cholera stools are the main, if not the only, channel of infection, and that the great cause of the propagation of cholera is the contamination, with the cholera stools, of the water used for drinking purposes. Milk may also be the vehicle by which it spreads. Flies may act as carriers of the contagion. It is claimed that the bacillus is inert in the intestinal canal unless the individual is in the "receptive state"—this is, the subject of intestinal catarrh, such as results from eating unripe fruit, and indigestible food, and beer and spirit drinking. It is also determined that the bacilli are destroyed by acids, and that if the stomach be normal, cholera will not result. "With pure water, pure air, pure soil, and pure habits, cholera need not be feared." (Hart.)

Little, if any, danger exists from being in the presence of the affected, although the emanations from the cholera excreta in the atmosphere may generate the disease if swallowed or inhaled. The dead bodies of cholera subjects apparently possess slight infective property, "the bacteria of decomposition" probably destroying the cholera germs. One attack does not afford protection against another.

The disease is usually observed during the summer months, and exempts no age. Debility, ill-health, gastrointestinal catarrh, fright, anxiety, fatigue, intemperance, and uncleanliness are predisposing causes. The incubation period is from three to five days.

Pathologic Anatomy.—The morbid appearances in the majority of cases of death from cholera may be thus summarized. The temperature generally rises after death, the body remaining warm for a considerable time. Rigor mortis rapidly ensues, the muscular contractions being often so powerful as to displace and distort the limbs. The skin is mottled and the body greatly shrunken. The blood is dark in color and thick. The arteries are empty of blood; the veins, on the other hand, are distended. The organs are, as a rule, pale and shrunken. The stomach and intestinal mucous membranes are congested, and present evidence of extravasation and ecchymoses, or are bleached and pale. The stomach and intestines usually contain a quantity of whey-like material, having an alkaline reaction, as well as quantities of cast-off epithelium and the bacillus. It is thought by many that the stripping-off of the epithelium is a post-mortem phenomenon. The Peyer's solitary and Brunner's glands are usually enlarged and prominent, and occasionally evidences of ulceration are apparent in the solitary glands, and sections placed under the microscope show the " comma bacillus." The villi of the mucous membrane, as well as the epithelium of the small intestines, are stripped off, leaving the basement membrane, for the most part, exposed. The liver is more or less advanced in fatty degeneration, presenting a somewhat mottled, yellowish discoloration. The kidneys are congested, the epithelium of the tubules granular, and detached from the basement membrane, blocking up the tubes. Bartholow observed, in all of his autopsies, "considerable hyperemia and dilatation of the vessels of the medulla oblongata. The constancy of this lesion would seem to indicate a relationship between congestion of the medulla and the cramps." The symptoms are in all probability induced by the absorption of poisons generated by the microorganisms in the intestinal tract.

Symptoms.—In accordance with the law of epidemic infectious diseases, the onset, course, and character of the symptoms vary in different cases and at different periods in the same epidemic.

The disease may either set in suddenly in a patient previously in good health, or it may follow an attack of rather severe and persistent diarrhea, with pain, nausea, vomiting, and depression. Such cases are termed *cholerine*, the stools of which are infections.

In a typical case, there are three stages: *first*, diarrhea; *second*, prostration; *third*, collapse, or, in favorable cases, reaction.

First Stage. This stage begins with chilliness, excessive thirst, coated tongue, unpleasant taste in the mouth, slight abdominal pain, and three or four copious, watery, yet fecal stools during the day, and a decided feeling of weakness, the stools rapidly becoming whey-like, easily voided, but with force and only slight pain. Occasionally an erythematous rash is present.

Second Stage. The stools rapidly increase in number, are voided with a rushing force, and consist of many quarts of grayish, or whitish, rice-water-like fluid, accompanied with forcible vomiting, first of the contents of the stomach, mixed with more or less bilious matter, afterward of the peculiar rice-water-like material; thirst becomes most intense, increasing or diminishing with the variations in the number of the stools and vomiting; severe muscular cramps soon follow, most severe in the calves, although occurring in all parts of the body.

Third stage. The stools, vomiting and cramps continue. The appearance of the patient becomes frightful; the eyes are sunken and surrounded by blackened rings, the nose pinched and pointed, the cheeks hollow, and the lips blue (facies cholerica); the surface cold and moistened with a sticky perspiration; the skin of the hands and fingers has the sodden appearance of the "washerwoman who has washed all day," and if picked up in folds, the fold but slowly disappears. The temperature rapidly falls, the pulse becomes small and compressible, barely perceptible at the wrist, and the heart-beats are scarcely recognizable. The voice is weak, husky, and sepulchral (vox cholerica), the tongue is like ice, the breath is cold and icy, the urine markedly diminished and albuminous. The mind is clear, but most patients are apathetic and indifferent to their danger. This, the algid state of cholera, or *cholera asphyxia,* usually terminates in death in from three to twelve, twenty-four, or forty-eight hours, but *reaction* may be established.

Stage of Reaction. The temperature of the body rises, the pulse gradually becomes fuller and stronger, the countenance becomes brighter, the stools less frequent and more fecal, the vomiting decreases, the thirst lessens; the urine increases in amount, but continues albuminous, the patient entering a slow convalescence, or typhoid symptoms develop, the so-called cholera typhoid, which prolongs the recovery for several weeks. Cases are sometimes observed in which collapse and death occur without any intestinal discharges. These are termed *cholera sicca.*

Convalescence is often prolonged and complicated by the development of severe bed-sores, boils, bronchitis, pneumonia or parotitis.

The prodromal stage lasts from a few hours to a week; the stage of collapse from a few hours to 12 or 24 hours; the stage of reaction a few hours, and the stage of convalescence several weeks.

Complications and Sequels. — Suppuration of the parotid gland; nephritis, painful tetanic contractions of the flexor muscles of the limbs, pneumonia, pleurisy, corneal ulcers, abscesses, ulcers, or gangrene of the extremities, profuse sweats, and various cutaneous eruptions.

Diagnosis.—The epidemic character, rapid spreading, and great mortality of the affection prevents its being mistaken for any other disease, although isolated cases are often confounded with cholerine or with cholera morbus, the points of distinction being very few.

The "comma bacillus," however, is present only in the discharge of true Asiatic cholera.

Prognosis.— Very unfavorable, the mortality ranging from twenty to eighty percent. The last epidemic in this country was much milder than former ones The prognosis is controlled by the general condition of the patient, the age, habits, and the development of the algid state; the prognosis being more favorable in those cases which develop gradually than in those in which it reaches its acme at a single bound; the very young or very old, those addicted to the various excesses and surrounded by unfavorable hygienic conditons, are more apt to perish than are others.

Treatment.—The treatment should always be instituted as early as possible, the arrest of the disease in the diarrheal stage being comparatively easy, while in the stage of collapse it is an exceptional occurrence.

The *prophylactic treatment* consists in isolation of the patient, sterilization of the discharges by chlorinated lime or carbolic acid, boiling of all bed-linen, napkins, towels, dishes, knives, etc., and in the event of death, wrapping the patient in sheets soaked in bichlorid of mercury solution (1 to 1000). Attendants on cholera patients should avoid direct communication with other individuals, and should be careful to thoroughly wash the hands after contact with the patient, and to protect the hair, the clothing, and the shoes by some covering that may be easly discarded. Non-infected individuals in cholera districts should be instructed to drink none but sterilized water and milk, and to partake only of light, easily digested food. The food supply should be protected from contamination by insects. Intemperance, overwork, excitement, and exposure to cold and wet should be avoided, and gastrointestinal disturbances should be guarded against. Immunization by means of protective serums has been practiced with some degree of success, but has not yet reached perfection. Haffkine's Serum

Medicinal Treatment.—The patient should be placed in bed at once as soon as the symptoms of diarrhea present themselves, and all food temporarily withheld. Calomel (beginning with two or three doses of gr. vii (0.45 Gm.), followed with small doses, gr. ¾ (0.048 Gm.), every two hours) is of value in the prodromal stage, especially if there is any indigestible food present in the gastrointestinal tract (Ziemssen). The opiates, mineral acids, and intestinal antiseptics are of great value in the early stage. The following formula, recommended by Bartholow, is productive of great benefit:

℞. Acid. sulphuric. aromat..fʒ v	20. Cc.
Tinct. opii deodorat.....fʒ iii	12. Cc.

M. S.—Ten to twenty drops in water every two hours.

Any of the mineral acids, hydrochloric, nitromuriatic, or sulphuric, in doses of ♏x-xv (0.66-1 Cc.), of the dilute acid are valuable, especially when combined with paregoric or laudanum. Squibb's cholera mixture may also be employed:

℞. Tr. opii,	
Spts. camphorae,	
Tr. capsici ....āā f℥ i	30. Cc.
Chloroformi pur. ......fʒ iii	12. Cc.
Alcohol ....q. s. ad f℥ v	150. Cc.

M. S.—Teaspoonful every two hours.

The formula of the "Sun Cholera Mixture" is:

℞. Tr. opii,	
Tr. capsici,	
Tr. rhei,	
Spt. camphoræ,	
Spt. menthæ pip. ....āā f℥ i	30. Cc.

M. S.—Teaspoonful in water after each evacuation of the bowels.

The following formula is also employed in this stage:

℞. Tr. opii,
Tr. capsici,
Tr. zingib,
Spt. menth. pip,
Spt. chloroformi,
Spt. camphoræ......āā fʒ ss 2. Cc.
Spt. vin. rect....q. s. ad f℥ ii 60. Cc.
M. S.—Teaspoonful in hot water every fifteen minutes until relief is afforded.

Intestinal antiseptics, such as bismuth, salol, lead acetate, etc., may be used with benefit. Peroxide of hydrogen internally has been used with success.

℞. Hydrogen peroxid. ....f℥ ii 60. Cc.
Aquæ destillat. ....f℥ viii 237. Cc.
M. S.—Cupful every two hours.

Enteroclysis or irrigation of the intestinal canal with large amounts, from one to three gallons twice daily, of hot, soaped water, hot four percent solutions of hydrogen peroxid, or weak solutions of tannin, or hot one percent solutions of common salt. The enteroclysis is accomplished by means of a soft rubber tube, one meter in length and of suitable size, to be introduced into the rectum, in front of the promonotory of the sacrum, into and up through the sigmoid flexure and into the descending colon. This tube, which is connected with a reservoir, should not be too small nor too large, in order to facilitate its introduction through the folds of the sigmoid portion of the lower bowel. In fact, the greatest difficulty to be encountered is to successfully pass the tube in front of the promontory of the sacrum, and enter it into the sigmoid flexure. The tube should be of proper firmness to prevent it from bending or buckling upon itself when the end (which in all cases should be rounded) comes in contact with the obstructing folds of the intestine.

In the second stage, the indications are to relieve the pain and cramps, check the discharges, and to support the patient. The distressing vomiting will call for the use of lavage of the stomach with hydrogen peroxid (two ounces to the quart of hot water), iced champagne, cocain, or hydrocyanic acid. Ice or carbonated waters may be given to allay the thirst. Morphin hypodermically is of greatest value in relieving the muscular cramps. Bartholow advises the following prescription:

℞.	Chloral ....ʒ iij	12.	Gm.
	Morphinæ sulph. ....gr. iv	.26	Gm.
	Aquæ lauro-cerasi .....f℥ j	30.	Cc.

M. S.—For hypodermic injection. Dose fifteen to thirty minims.

Locally, hot applications, hot irons, hot bricks, hot water-bottles, etc., or an ointment of chloroform or chloral will be of service. Inhalatins of chloroform or ether may be necessary. Brandy, whiskey, ammonia, strychnin, etc., should be administered to sustain the patient. Subcutaneous, intravenous, and rectal injections of hot, normal salt solution (teaspoonful of salt to quart of water) are necessary to compensate for the fluid lost by the discharges. The astringent rectal irrigations should be continued. When the patient becomes algid, hot baths and hot applications should be employed.

In the stage of reaction, feeding is renewed, peptonized milk, milk and lime water, gruels and similar liquid foods being allowed. Tonics, such as iron, arsenic, quinin, etc., should be given.

TETANUS.

Synonyms.—Lockjaw; trismus; cephalic tetanus.

Definition.—An acute or subacute infectious disease, characterized by muscular rigidity, with paroxysms of tonic convulsions, the mind remaining clear.

Idiopathic tetanus when no open wound is discoverable.

Traumatic tetanus when an open wound is present.

Tetanus neonatorum when it attacks infants.

Lockjaw or *trismus* when the jaw alone is involved.

Cephalic tetanus when the throat and face are affected.

Cause.—The result of a specific bacillus, which usually gains access to the system through an abrasion. The incubation period is from a few days to four weeks.

Pathologic Anatomy.—In the post-mortem examinations which have been made, no uniform morbid appearance was discovered on microscopic examination. The brain, cord, lungs, and muscles are markedly congested, and show minute hemmorrhages, such as are met with in all cases of death from convulsions, and which occur chiefly during the process of death. In four post-mortem examinations of cases dying from tetanus at the Philadelphia Hospital, marked chronic nephritis was observed. Probably the future may show some connection between nephritis and tetanus, by which the specific poison is not eliminated as it might be were the kidneys normal.

Symptoms.—The onset is rather sudden, with stiffness of the jaw, neck, and tongue, and some difficulty in swallowing, which increases in extent, the stiffness passing down the spinal muscles to the legs, which are held in a firm spasm. Gradually tonic spasms develop, which, involving the jaw muscles, cause "lockjaw;" the face muscles, "risus sardonicus;" neck and trunk muscles, "opisthotonos;" these tonic convulsions are associated with intense pain, and the patient suffers the greatest distress, particularly if the chest muscles are involved. Usually the febrile reaction is slight, but in many cases 102° F. to 104° F. is reached, and in some instances, as death approaches, 108° F. to 110° F., rising still higher after death. The mind remains clear till carbonic acid poisoning occurs. Usually a wound, not severe, can be found, the symptoms developing some two weeks after its occurrence. The tonic spasms are developed by many sources of irritation, a draught of air, shaking of the bed or floor, suddenly opening the door of the room, the presence of a visitor, or attempts at speaking or movement.

Diagnosis.—The symptoms are so characteristic, with the addition of a history of a wound, that an error seems hardly probable.

Tetany. The spasms chiefly affect the extremities, the muscles being free in the interval and trismus a late or very rare condition.

Strychnin poisoning often closely resembles tetanus, but there is no beginning trismus and more rapid development of the symptoms. No history of wound.

Hydrophobia does not have trismus, but respiratory spasm, excited by attempts at swallowing, with increasing mental symptoms.

Prognosis.—Unfavorable. The great majority die.

Treatment.—The patient should be placed at absolute rest in bed in a quiet and darkened room. The wound should be opened and antisepticized. The spasms will require the administration of drugs such as chloral, potassium bromid, chloralamid, morphin sulphate, physostigma, and antipyrin. Inhalations of chloroform or amyl nitrite are often necessary. The administration of tetanus antitoxin by subcutaneous injection has been followed by successful results in a number of cases. Nutrition must be maintained by rectal alimentation. The hypodermic injection of carbolic acid, gr. iii (0.2 Gm.) a day, increasing rapidly until gr. vi-viii (0.4-0.5 Gm.) daily is reached, has been highly recommended. Bacelli employs a two percent solution hypodermically every three hours.

HYDROPHOBIA.

Synonym.—Rabies.

Definition.—An acute infectious disease, occurring in the lower animals, but communicable to man by inoculation, characterized by intense tonic spasm beginning in the larynx.

Cause.—The disease is due to a specific virus which gains entrance to the general circulation of man by means of the bites of rabid animals. The poison is contained in the medulla, brain, and secretions, especially the saliva. Various organisms have been found, but their connection with the disease is far from proved. The affection in man is usually contracted through the bite of a rabid dog. However, not more than 15 percent of those bitten by dogs become affected (Horseley). Bites on the hands and face are especially liable to be infected by the virus. The period of incubation varies from one week to two or more months. If no symptoms manifest themselves within three months, the patient may be considered as unlikely to develop the disease.

Pathologic Anatomy.—The structural changes are confined to the upper spinal cord, medulla, pons, and cerebral cortex. The blood vessels are dilated and overfilled, the perivascular sheaths are infiltrated with leukocytes, and small hemorrhages are present. The ganglia of the cerebrospinal and sympathetic systems undergo characteristic changes. The capsular cells of the ganglia proliferate leading to destruction of the ganglia, with their replacement by round cells. Occasionally the ganglion cells are but slightly altered. The pharynx, larynx, trachea, and bronchi are hyperemic.

Symptoms.—The first stage lasts about twenty-four hours and begins with pain in the wound or its cicatrix, depression of spirits, irritability, mental anxiety, feverishness, anorexia, hoarseness, sleeplessness, and hypersensitiveness to noises. This is followed by the second, spasmodic, or furious stage. The muscles of the larynx become extremely irritable and contract on the slightest excitation, thus rendering swallowing and breathing difficult. Any attempt to swallow water or the accumulations of saliva induces the paroxysms. Hyperesthesia is marked, so much so that even a breath of air or a slight noise may cause the spasms. Delirium and maniacal excitement are often present. Fever (103° F.) is observed also in this stage, and the pulse is irregular. The duration is from one to three days. In the third stage prostration becomes marked and the paroxysms subside. The heart gradually fails. Death follows from syncope, or asphyxia from convulsions. This stage lasts from six to eighteen hours.

Diagnosis.—*Tetanus* is distinguished from hydrophobia by its history, incubation period, character of convulsions, and absence of marked throat symptoms.

Hysteria in persons bitten by animals may simulate hydrophobia. Such individuals are usually neurotic and attempt to bark and bite and show many manifestations uncommon in hydrophobia. The resemblance is often very close. Subdural injections in rabbits, of the central nervous system of the animal supposed to be rabid, will, in true hydrophobia, be followed by the paralytic form of the disease in fifteen to twenty days.

Prognosis.—After the disease is established the prognosis is extremely unfavorable. Nearly all cases die, very exceptionally spontaneous recovery occurs.

Treatment.—Prompt cauterization of the wound with some strong caustic or the actual cautery is advised. The wound should be kept open. Chloroform, chloral, opium, etc., will be necessary to control the spasms. Nutritive enemas will be necessary to keep up the patient's strength. The Pasteur treatment, consisting in a series of inoculations of virus, of increasing strengths, prepared from the spinal cords of infected rabbits, is worthy of a trial for immunizing and curative purposes. The individual bitten by a rabid animal should receive this treatment immediately. The treatment is harmless to a non-infected person (Tyson).

WHOOPING COUGH.

Synonyms.—Hooping cough; pertussis.

Definition.—An infectious disease characterized by a convulsive paroxysmal cough, consisting of a number of forcible expirations, followed by a series of deep, loud, sonorous inspirations (the whoop), repeated several times during each paroxysm, and associated with catarrh of the bronchial tubes.

Causes.—The disease is contagious and is due to an unknown microörganism associated with the sputum. It is a disease of childhood, fully one-half of the cases occur during the first two years of life. Adults may be affected. One attack usually secures immunity.

Pathology.—There are no characteristic structural changes. The poison which produces the disease acts on the nervous system and respiratory mucous membrane. It is said that "irritation of the internal branch of the superior laryngeal nerve produces relaxation of the diaphragm, spasm of the glottis, and a convulsive expiration, the series of phenomena present in a paroxysm of asthma." There is also hyperemia of the mucous membrane of the nares, pharynx, larynx, and bronchial tubes, with diminished secretion, followed by an increased secretion of a transparent mucus, afterward becoming purulent, the mucous membrane pale and anemic. Fatal cases are nearly always due to extension and exaggeration of this congestive condition.

Symptoms.—These may be considered in three stages: catarrhal, spasmodic, and terminal.

Catarrhal stage originates in an ordinary naso-laryngo-bronchial catarrh, with a loose cough. Duration, one or two weeks.

Spasmodic Stage: The cough becomes *paroxysmal*, consisting of a succession of short, rapid, expiratory efforts, the face becoming red, the eyes swollen and protruding, the body bending forward, and when these expiratory efforts have exhausted the breath, they are followed by a deep, loud, crowing inspiration—the whoop: each paroxysm being composed of three such spells, the last one followed by the expectoration of a small amount of tough, viscid mucus. The attacks of cough may be so severe as to cause vomiting, and if the vomiting occur shortly after food has been taken, the nutrition of the patient will suffer. Profuse epistaxis is not infrequent. Duration, about four weeks.

Terminal Stage: The paroxysms recur at longer intervals, are of shorter duration and less intensity, the catarrhal symptoms being more marked, the expectoration freer. Duration, one or two weeks, often followed by the "cough of habit."

Complications.—The most common complications are congestion of the lungs, capillary bronchitis, pneumonia, emphysema, and collapse of the lung. Convulsions, hydrocephalus, and apoplexy may occasionally occur as complications.

Diagnosis.—This is certain only during the second or paroxysmal stage, the "whoop" of which is especially characteristic and distinctive.

Prognosis.—Depends upon the age and strength of the patient, the severity of the paroxysms, and the presence or absence of complications. Ordinary cases are favorable. Moderately severe attacks during infancy are followed by cerebral symptoms, while attacks occurring in adults are followed by chest symptoms.

Treatment.—There is no specific treatment. The disease is self-limited. The symptoms may be modified by treatment and complications avoided. Isolation of the patient and disinfection of all his personal articles should never be neglected. The patient need not be confined to bed. A well-ventilated room, with plenty of sunlight, should be selected for the patient. On nice days he should be allowed in the open air as much as possible, being careful to protect him from catching cold by the wearing of warm clothing. The diet should be nutritious, but should be regulated to the individual. Inhalations of creosote or eucalyptol are very valuable, dropped upon cotton in a respirator, or vaporized over an alcoholic lamp, or cloths dipped in solutions of these drugs may be hung about the room.

The medicinal treatment includes a number of remedies. Da Costa recommends quinin sulphate, in full doses, and chloral alone or combined with the bromids. He also employs a spray of sodium bromid, gr. xx (1.3 Gm.), fluid extract of belladonna, ♏ ii (0.12 Cc.), and water, f℥i (30 Cc.). Ammonium bromid may also be used. At times benefit may be obtained from the administration of antipyrin, gr. ⅙-v (0.011-0.3 Gm.), acetanilid, gr. i-iii (0.065-02 Gm.) every four hours, or phenacetin, gr. i-ii (0.065-0.13 Gm.)

four times daily. These are most efficacious when administered in expectorant mixtures. Holt advises the use of antipyrin in one grain (0.065 Gm.) doses every three hours for a child six months old. Terpin hydrate, gr. i-v (0.065-0.3 Gm.) is sometimes valuable. Belladonna may be added to any of the remedies named with advantage, or the tincture may be used alone in doses of ♏v-x (0.3-0.6 Cc.) three times daily, gradually ascending, until flushing of the surface is observed, after which the dose is continued that maintains the flushing.

Starr recommends the following for a child of one year:

℞.	Ext. belladonnæ	gr. j		.065 Gm.
	Aluminis	ʒ ss		2. Gm.
	Syr. zingiberis,			
	Syr. acaciæ,			
	Aquæ	āā f℥ j	āā	30. Cc.

M. S.—A teaspoonful four times in the twenty-four hours.

During convalescence tonics should be administered. Cod-liver oil, quinin, iron, etc., are of great value in this period in preventing pulmonary sequels.

DISEASES OF THE DIGESTIVE TRACT.

DISEASES OF THE MOUTH.

Introduction.—The angles of the mouth may be seats of radiating scars due to old syphilitic cracks or fissures. Herpes or fever blisters in the same situation may lead to more or less confusion, but the duration and absence of scarring will soon render the diagnosis clear. The inside of the lips, the buccal mucous membrane, and the tonsils may be affected by the initial lesion of syphilis, the chancer, and in all indurated lesions in these situations this disease should be carefully considered. Mucous patches or moist papules are common on the mucous membrane of the mouth and their importance arises from their contagious nature. Inflammation of the gums, or gingivitis, is a rare condition which may arise from gonorrheal infection, mercurial-poisoning, scurvy, and other similar constitutional diseases. A blue line on the gums, near the insertion of the teeth, is indicative of lead-poisoning. First dentition is usually completed at the end of the second year, and the permanent teeth begin to appear in the fifth or sixth year, any delay in dentition or the eruption of badly-formed teeth is attributed to nutritional disorders, such as occur in rickets and syphilis. Hutchinson's teeth consist in certain characteristic alterations in the permanent teeth, and indicate congenital syphilis. The lateral incisors are peg-shaped, and the central incisors have convex sides and notched cutting edges. Carious teeth may arise from uncleanliness of the mouth, drugs, and nutritional disturbances.

CATARRHAL STOMATITIS.

Synonyms.— Simple stomatitis; erythematous stomatitis; catarrh of the mouth.

Definition.—An acute catarrhal inflammation of the whole or a portion of the mucous membrane of the mouth and tongue, characterized by pain, redness, swelling, restlessness, slight fever, fetor of the breath, and disordered secretion. It is most common in infants and children. It results from the introduction of irritants into the mouth, gastrointestinal disturbances, delayed dentition, and the infectious fevers. Chronic stomatitis occurs mostly in adults as the result of alcoholic or tobacco excesses.

Treatment.—The most important point in the treatment is the removal of the exciting cause, attention to the secretions and diet, and gently mopping out the mouth at frequent intervals with a soft wad of absorbent cotton and cold or iced water, or diluted Dobell's solution (see Scarlet Fever), or the following:

℞. Sodii boratis	gr. xc	6. Gm.
Aquæ destillat.	f℥ iss	45. Cc.
Mel. rosæ	f℥ iss	45. Cc.

M. S.—Mouth wash.

In severe or aggravated cases a dilute solution of silver nitrate, gr. ij-v (0.13-0.32 Gm.), to f℥j (30 Cc.), should be applied.

APHTHOUS STOMATITIS.

Synonyms.—Follicular stomatitis; vesicular stomatitis; croupous stomatitis.

Definition.—An acute inflammation of the follicles and mucous membrane of the mouth and tongue, characterized by a fibrinous or croupous exudation; the exudation first appearing in isolated spots (*aphthæ discrete*), afterward coalescing, and forming large and irregular sized patches (*aphthæ confluens*), which rupture, leaving an ulcer, which slowly heals. The disease occurs mostly in childhood and is due to difficult dentition, disorders of digestion, uncleanliness, and the eruptive fevers. The lesions appear usually as small white vesicles which subsequently rupture. Pain, difficulty in swallowing, salivation, feverishness, and fetor of the breath are present.

Treatment.—The exciting cause should be removed if possible. The mouth should be cleansed after each feeding, and nursing bottle and nipples should be sterilized by boiling. Digestive disturbances should be corrected by the administration of powders containing calomel, gr. 1-12 (0.005 Gm.), and sodium bicarbonate, gr. i (0.065 Gm.) every three hours. Small doses of quinin sulphate may be necessary in protracted cases. Locally, chlorate of potassium or boric acid in solution will be of benefit. The ulcers may be touched with a weak solution of silver nitrate (gr. iv to f℥i). Honey and borax—the *mel boracis* of the U. S. Pharmocopea is also efficacious.

ULCERATIVE STOMATITIS.

Synonyms.—Diphtheritic stomatitis; gingivitis ulcerosa.

Definition.—An acute diphtheritic inflammation of the mucous membrane of the mouth, which terminates in ulceration.

Causes.—It is probably infectious in character as it is usually seen in epidemics. It occurs with greatest frequency in the families of the poor, in institutions, jails, and camps, and in those reduced in vitality by acute diseases, unhygienic surroundings, and poor food.

Symptoms.—It begins with swelling of the mucous membrane about the base of the teeth and adherent deposits appear on the gums, which eventually become gray or black and separate as sloughs, leaving behind irregular ulcers. The lower jaw is most often affected, but it may extend to the lips, cheeks, or tongue. The submaxillary glands are swollen and tender. Pain is present; mastication and deglutition are difficult; the mouth is hot; the saliva dribbles away mixed with blood and pulpy matter; the breath is fetid; anorexia is present; the patient is feverish and restless; and the gastrointestinal tract is disordered in its functions.

Prognosis.—A favorable termination may be expected when the affection is promptly and properly treated and when the ulcerated surface is not too extensive.

Treatment.—The exciting and contributory causes should be immediately removed. Internally potassium chlorate, gr. i-5 (0.065-0.32 Gm.) should be administered in solution every three hours. Iron, quinin, strychnin, and alcohol may be necessary if there is much depression. Locally, potassium chlorate solution, bismuth, alum, silver nitrate solution, or the following may be applied:

℞.	Cupri sulphat. ....gr. x	.65	Gm.
	Pulv. cinchonæ opt......gr. cxx	8.	Gm.
	Pulv. g. arab............gr. lx	4.	Gm.
	Mel. commun. ....fʒ ij	8.	Cc.
	Aquæ font. ....f℥ iij	90.	Cc.

M. S.—The ulceration to be touched twice daily with the point of a camel's hair pencil. (Dewees).

THRUSH.

Synonyms.—Parasitic stomatitis; muguet; sprue; white mouth.

Definition.—An inflammation of the mucous membrane of the mouth, associated with or caused by the growth of a parasitic plant, the *Oïdium albicans;* characterized by pain, disorders of digestion and of the bowels. It is most common in bottle-fed infants, but may be seen in adults in the last stages of cancer or consumption. The mucous membrane of the mouth presents a dark red appearance in isolated patches on which whitish points appear and rapidly coalesce. These whitish points are made up of epithelium, fat, and fungus, and resemble curdled milk. The symptoms common to the other forms of stomatitis are also present.

Prognosis.—The termination is favorable under treatment in all cases except those due to malignant diseases.

Treatment.—Absolute cleanliness as regards the baby's mouth, nursing bottles, nipples, etc., is necessary. The disordered digestive tract should receive attention, largely by the proper modification of the milk. A saturated solution of sodium hyposulphite, ♏iii-x (0.18-0.6 Cc.), may be given internally and also applied locally. Sodium bicarbonate, and sodium biborate may also be employed locally.

℞.	Sodii boratis	gr. lx	4. Gm.
	Glycerini	fʒ ij	8. Cc.
	Aquæ	fʒ vj	24. Cc.

M. S.—To be thoroughly applied four or five times daily, and continued for a week after the disappearance of the affection.

GANGRENOUS STOMATITIS.

Synonyms.—Cancrum oris; noma; water-cancer.

Definition.—An acute, rapidly progressive gangrenous ulceration of the mouth, leading to extensive sloughing and destruction of the affected tissues.

Causes.—It is probable that gangrenous stomatitis is due to some parasitic microörganism, but its character is as yet unknown. It attacks feebly and sickly children by preference; and is occasionally observed in adults. It may occur as a primary affection, but is most often encountered as a sequel to measles, scarlet fever, typhoid fever, or pneumonia.

Symptoms.—All the symptoms common to the other varieties of stomatitis are present to a marked degree. One of the first manifestations is the penetrating gangrenous odor. The cheek is swollen and edematous, and the skin has a glazed, waxy appearance. On eversion of the cheek a foul sloughing ulcer is brought into view, which shortly perforates the skin and discharges externally. Constitutional reaction is severe; the temperature is high and irregular; the pulse rapid and feeble; and prostration is marked.

Diarrhea is common. Death usually terminates the affection in a week to ten days. Recovery is very rare.

Treatment.—Eversion of the cheek with cauterization of the ulcer by stick silver nitrate, fuming nitric acid, or the Paquelin cautery is the first indication. The mouth should then be kept as clean as possible by means of peroxid of hydrogen, boric acid solution, or other mild antiseptics. The strength of the patient should be maintained by the administration of nourishing food, whiskey or brandy, quinin, iron, strychnin, and other tonics.

MERCURIAL STOMATITIS.

The ingestion and absorption of mercurial preparations in excess of their physiologic dose or in susceptible individuals induce tenderness of the gums, fetor of the breath, metallic taste, increase in saliva, and redness of the mucous membrane of the mouth. In marked cases salivation is profuse, the tongue is swollen and protrudes from the mouth, and necrosis of the teeth and jaw occurs.

Treatment.—The mercurial preparations should be immediately suspended and potassium iodid administered in small doses. A saturated solution of potassium chlorate should be employed as a mouth wash. Atropin may be given to control the excessive secretion of saliva. Tonics are often necessary to combat the anemia.

ANGINA LUDOVICI.

Synonyms.—Ludwig's Angina; cellulitis of the neck; cyanche gangrenosa.

Definition and Symptoms.—A phlegmonous inflammation of the floor of the mouth and tissues about the side of the neck. It is probably a streptococcus infection and is observed after the infectious fevers, traumatism, and dental caries. It may end in suppuration, gangrene, septicemia, and very rarely resolution. Pain, swelling, dysphagia, dyspnea, and grave constitutional symptoms are present.

Treatment.—Tonics and stimulants internally and ice and leeching locally are indicated. Surgical interference is usually necessary.

DISEASES OF THE TONGUE.

Coating of the Tongue.—Normally, the color of the anterior two-thirds of the tongue is a pale red, while the posterior third is grayish. On the anterior portion are seen the fungiform papillae as bright red points, and on the posterior portion the circumvallate papillae arranged in two rows of red circles. Fur on the tongue is due to accumulated epithelium, fungi, and food particles. It is uniform in febrile diseases, gastrointestinal disorders, naso-pharyngeal affections, and not uncommonly in health. A circumscribed furring usually points to some local oral trouble. Unilateral furring results from some disturbance of the second and third branches of the fifth nerve. Localized thickenings of the epithelium of the tongue give to it a chart-like appearance to which the term *geographic tongue* is applied. Intense white spots on the mucous membrane constitute *leukoplakia*. The *pale tongue* is noticed in anemia. The *dry, brown,* and *fissured tongue* accompanies the low fevers, such as typhoid fever and dysentery. The *black tongue* may be parasitic in nature, but is usually observed in malignant fevers. A *bluish-black tongue* is occasionally seen in Addison's disease. The *red, beefy tongue* is most often encountered in diabetes and similar wasting diseases. The *strawberry tongue* consists of a more or less uniform whitish coating, through which project the bright red fungiform papillæ. It is seen in scarlet fever. *Trembling tongue* may be seen in paresis and similar nervous diseases, alcoholism, and asthenic fevers.

GLOSSITIS.

Definition.—An inflammation of the parenchyma of the tongue; characterized by great swelling of the organ, with difficult mastication, deglutition, and vocalization. It may be acute or chronic. The affection may be due to injury, contact with boiling liquids or other irritating substances, or stings of insects.

Symptoms.—The tongue is swollen, painful, and sometimes protrudes from the mouth, thus interfering with mastication and deglutition. The discomfort is extreme. The voice is muffled and there may be dyspnea. There is increased flow of saliva. Fever and other constitutional phenomena are present. Suppuration may occur. Acute glossitis usually terminates in recovery within a week, although death may occur from suffocation. Chronic glossitis persists indefinitely being manifested largely by pain aggravated by movements of the tongue.

Treatment.—The application of ice to the tongue and to the jaw affords relief in most cases. Occasionally heat must be substituted. Deep scarification is necessary in aggravated cases. Suppuration indicates prompt incision. Antiseptic mouth washes should be employed constantly. In chronic glossitis, silver nitrate in stick or solution should be applied to ulcerated areas. The constitutional phenomena should be treated on general principles. If suffocation appears imminent tracheotomy must be performed.

SYPHILIS OF THE TONGUE.

Syphilis of the tongue may appear as the chancer, mucous patch, or gumma. The characteristics of these lesions are maintained in this structure. The chancer is distinguished largely by its parchment-like induration, and the age at which it appears. The mucous patch is usually associated with other signs of secondary syphilis and the gumma is diagnosed by exclusion and the therapeutic test.

Treatment.—Mercury and the idodids, alone or combined, should be administered over an extended period.

ULCERATION OF THE TONGUE.

Simple ulceration may result from carious teeth, gastrointestinal irritation, and contact with irritants. The underlying causes should be removed and the ulcerated areas touched with silver nitrate in stick form or in solution.

Tuberculous ulceration is rare and appears on the dorsum, near the tip of the tongue, as an irregularly oval ulcer with undermined edges and an uneven base, which is covered with coarse pinkish-gray granualtions. It is incurable. The general treatment for tuberculosis may be employed, supplemented by the use of the X-ray.

Malignant ulceration is usually due to epithelioma, and occurs with greatest frequency in men past 40 years of age. The lesion has hard and everted edges, with an uneven, excavated base. The adjacent tissues are infiltrated and indurated, and the neighboring glands are involved. Neuralgic pain is constant. Removal of the organ is indicated. The affection terminates fatally. Radiotherapy may be of benefit.

DISEASES OF THE PHARYNX.

ACUTE CATARRHAL PHARYNGITIS.

Synonyms.—Angina catarrhalis; acute "sore throat."

Definition.—An acute catarrhal inflammation of the mucous membrane of the tonsils, uvula, soft palate, and pharynx; characterized by rigors, fever, painful deglutition, coughing, or constant desire to clear the throat, with a more or less decided nasal intonation of the voice.

Causes.—Exposure to cold and wet, infective microörganisms, local irritants, such as hot liquids and noxious gases, rheumatism, gout, and the eruptive fevers may give rise to acute pharyngitis.

Symptoms.—The onset is sudden, usually with rigors followed by fever, thirst, headache, anorexia, coated tongue, foul breath, dryness of the throat, painful deglutition, and a constant desire to clear the throat, due to the increased length of the uvula. Extension to the eustachian tube and middle ear gives rise to deafness and earache. The nasal tone of the voice is almost pathognomonic. Inspection of the pharynx reveals an intensely red and swollen condition of the mucous membrane. The tonsils and larynx may also be involved. Secretions are first lessened, but soon become increased and assume a thick, tenacious, opaque character.

Varieties.—*Exanthematous pharyngitis* is the form of the affection complicating the acute infectious diseases, such as scarlatina, measles, influenza, and small-pox.

Erysipelatous pharyngitis is the form complicating facial erysipelas; rarely, however, the affection begins in the pharynx, spreading to the face and other parts. The association of nasal inflammation with erysipelas should not be forgotten.

Gangrenous pharyngitis may occur with diphtheria, scarlatina, erysipelas, small-pox, and typhoid fever. The symptoms assume a typhoid (depressed) character, the termination being usually fatal.

Phlegmonous pharyngitis is the variety associated with an accumulation of pus in the submucous and deeper tissues of the pharynx, constituting a *retropharyngeal abscess.* This variety of pharyngitis may follow the penetration of a sharp piece of bone or be secondary to caries of the cervical vertebræ.

Fibrinous pharyngitis, or, as it is sometimes termed, *pseudomembranous* is observed in croup and diphtheria, under which headings it is considered.

Prognosis.—Favorable. The affection terminates in three or four days by the discharge of a quantity of thick opaque mucus.

Treatment.—Cases resulting from exposure are benefitted by the application of bicarbonate of soda by insufflation. Opium alone or combined with ipecac or camphor will often abort an attack. Pain may be relieved by the administration of salol, gr. iii (0.2 Gm.), phenacetin, gr. iii (0.2 Gm.), and powdered camphor, gr. i (0.65 Gm.), four to six times daily. Sodium salicylate, gr. x-xv (0.6-1 Gm.) every hour, for six hours, is of great benefit. Drop doses of tincture of aconite may be given to control the fever. Nuclein (Aulde), gr. i-iii (0.065-0.2 Gm.) every hour or two, has been followed by excellent results. The bowels should always be freely opened early in the treatment.

Locally, the application of a four percent solution of cocain or cocain lozenges will afford considerable relief. Ice pellets in the mouth and heat or cold applied externally also produce benefit. Gargels or sprays or alum (gr. viii to f℥i), ammonium chlorid (gr. xx to f℥i), or potassium chlorate (gr. x to f℥i) often relieve the swelling and congestion.

The severe cases are nearly always secondary affections, and their treatment is that of the primary diseases.

CHRONIC PHARYNGITIS.

Chronic inflammation of the pharynx follows repeated acute attacks, prolonged irritation, long-continued, overuse and improper use of the voice, chronic rhinitis, and digestive disturbances. It is common in hucksters, public speakers, singers, and smokers. In the early stage the mucous membrane is in a state of chronic hyperemia, and is thick, swollen, and studded with distended follicles and enlarged lymphatic glandules, which give to it a granular appearance. In the later stage the mucous membrane undergoes atrophic changes, and is anemic, glossy, and dry. As a result of these changes the voice is husky, and the throat is dry. Distress follows the use of the voice and there is a constant desire to clear the throat.

Treatment.—The underlying causes should be promptly removed. Tonics, such as iron, quinin, strychnin, and cod-liver oil, together with plenty of fresh air, should be prescribed. Locally, Dobell's solution and similar antiseptic solutions should be used in the pharynx and nose. When the granules are present, astringent applications, such as zinc sulphate (gr. v to f℥i) and silver nitrate (gr. x-xx to f℥i), or the galvanocautery may be employed. A spray, consisting of menthol (gr. ii), eucalyptol (gr. i), and liquid vaselin will be productive of great relief.

ULCERATION OF THE PHARYNX.

Ulceration of the pharynx seldom follows simple chronic pharyngitis, but results from syphilis, tuberculosis, diphtheria, typhoid fever, or scarlet fever. The history, character of the ulceration, and reaction to treatment will aid greatly in making a diagnosis. The treatment is largely that of the primary disease, but locally, in all, mild antiseptic and stimulating applications should be made to the ulcerated areas.

ACUTE TONSILITIS.

Synonyms.—Amygdalitis; quinsy; phlegmonous pharyngitis.

Definition.—An acute parenchymatous inflammation of one or both tonsils, with a strong tendency toward suppuration.

Causes.—The affection is most common in youth and early adult life, and is greatly influenced by rheumatic diathesis, exposure to cold and wet, inhalation of foul air, and previous attacks.

Symptoms.—The onset is more or less sudden with rigors, rise of temperature, 102° to 104° F., full frequent pulse, 100 to 120, headache, thirst, pain and swelling at the angle of the jaw, difficult deglutition, difficult breathing, muffling of the voice, and often impaired hearing and earache. Inspection reveals marked swelling and congestion of the mucous membrane of the fauces and pharynx. One or both tonsils will be seen to be enormously swollen and projecting towards the median line. The surface is covered with small, yellowish points which closely resemble patches of false membrane, but close examination will show them to be distended follicles from which cheesy, foul-smelling, pellets may be expelled.

If suppuration is imminent, the throat becomes more painful and throbbing in character, the constitutional reaction becomes more severe, and fluctuation may be obtained. Breathing is extremely difficult and relief is afforded when rupture occurs either spontaneously or as the result of a sudden effort at coughly or vomiting.

The disease lasts from three to seven days, terminating in resolution or in suppuration.

Diagnosis.—This is usually not difficult, but cultures should be made in doubtful cases.

Prognosis.—As a rule the disease ends favorably. Suffocation may occasionally occur, especially in weak children.

Treatment.—Rest in bed and liquid diet are the first indications. Calomel, gr. v (0.3 Gm.), and sodium bicarbonate, gr. v (0.3 Gm.), should be administered immediately, followed in six or eight hours by a saline cathartic. Sodium salicylate, gr. x-xv (0.6-1 Gm.) or cinchonidin salicylate, gr. v (0.3 Gm.) should then be given every two hours until six dozes have been taken. If the febrile reaction is very great tincture of aconite may be employed in very small doses, but if it is contraindicated internally, for any reason, it may be diluted with glycerin and painted over the affected parts. In advanced cases the following will be found of value:

℞. Tincture ferri chloridi . . fʒ ij	8. Cc.
Glycerini . . . . ad f℥ ij	ad 60. Cc.

M. S.—Teaspoonful every two hours, *undiluted*. Not to be followed by food for 1 hour.

Among the other remedies useful in this condition are the ammoniated tincture of guaiac, sodium benzoate, salol and phenacetius, and nuclein (Aulde). Ice pellets in the mouth will usually afford great relief. Opium may be necessary at times.

Locally, ice or heat may be applied to the angles of the jaw. The mouth should be kept as clean as possible by means of Dobell's solution and peroxid of hydrogen. Painting of the tonsils with nitrate of silver solution (gr. xl to f℥i) is recommended. Scarification of the diseased structures is sometimes very beneficial. The application of a solution of cocain (10 percent) may be of benefit. The occurrence of suppuration will necessitate the employment of hot applications and early incision, preferably at the upper and outer side of the gland, near the soft palate. When the acute symptoms have subsided, copper sulphate solution (gr. xx to f℥i), or Monsel's solution diluted (fʒi to f℥i) should be applied to hasten shrinkage of the glands.

HYPERTROPHY OF THE TONSILS.

Causes.—Enlargement of the tonsils may occur as the result of repeated acute attacks of inflammation, but may arise independently. It is most common in children. It may consist of hypertrophy of the glandular structure itself, the connective-tissue, or both. The consistency depends on the quantity of fibrous tissue present. Catarrhal inflammation and adenoid growths of the naso-pharynx are common accompaniments.

Symptoms.—Enlarged tonsils are always predisposed to inflammation and may remain unnoticed until such a condition arises. Inquiry will elicit the information that the patient breaths almost constantly with the mouth open, snores during sleep, is subject to night-terrors, has difficulty in swallowing, and is mentally dull. The voice is usually thick and of a nasal quality, hearing is impaired, and the face has a stupid expression. Development may be interfered with, resulting in narrowing of the anterior nares, contraction of the superior dental arch, elevation of the hard palate, and the formation of the "chicken-breast," so-called, the round or barrel chest, and the funnel breast. There is fetor of the breath and impairment of the special senses. Cough and stuttering are rather common.

Treatment.—The enlarged glands and any pharyngeal adenoids should be removed by a surgical operation, after which measures should be employed to correst the faulty development. Fresh air, exercise, proper diet, tonics, etc., will be of great benefit.

DISEASES OF THE ESOPHAGUS.

ESOPHAGITIS.

Acute inflammation of the esophagus may result from the swallowing of corrosive liquids, lodgment of foreign bodies, diphtheria, and small-pox. Chronic inflammation of the esophagus results from venous obstruction, such as follows valvular heart-disease and cirrhosis of the liver.

Symptoms.—The principal manifestations are pain beneath the sternum and difficulty in swallowing. There is a copious mucoid secretion which may be regurgitated or passed into the stomach. In acute cases, the resulting cicatricial changes eventually lead to obstruction.

Treatment.— Nothing can be done to aid in the cure of the local condition. Demulcents, ice, and liquid diet may be employed, but if deglutition is painful, it is best to resort to rectal feeding.

ESOPHAGEAL OBSTRUCTION.

Functional obstruction of the esophagus or *esŏphagismus* is an hysterical condition which is most frequently observed in women past middle life. In may occur also in chorea, epilepsy, and hydrophobia. Male hypochondriacs are sometimes affected. The condition is manifested by difficulty in swallowing, which is spasmodic in character, choking, and regurgitation of food. It may be excited by liquid as well as solid food. It is distinguishel from other conditions of the esophagus by the paroxysmal character of the obstruction, the absence of emaciation, the history, age, and sex of the patient, and the ease with which a bougie is passed.

Treatment.—The systematic passage of the esophageal bougie combined with approprite measures for the relief of the underlying neurotic condition generally results in cure. Care must be taken not to produce ulceration by the too-frequent employment of instrumental means.

Organic obstruction of the esophagus may be due to the presence of a foreign body in the lumen of the tube, a contracting cicatrix such as follows ulceration, acute esophagitis, tumors of the esophageal wall, such as cancer and rarely polyps, and external tumors, including aneurysm, enlarged lymphatic glands, and mediastinal growths. It is manifested by slowly increasing dysphagia, which is in turn followed either by regurgitation of the food or dilatation of the esophagus above the point of obstruction. Pain and emaciation are constant, and it is impossible to pass a bougie. A discharge of blood and mucus is common, after such attempts in cancerous obstruction. Death is nearly always the termination from starvation or exhaustion.

Treatment.—Rectal alimentation will be necessary to support the patient and opium may be required to relieve pain. In cicatricial obstruction bougies may be employed, but in other forms surgical treatment will be required. The outlook is unfavorable except in cicatricial obstruction.

DISEASES OF THE STOMACH.

DIAGNOSTIC TECHNIC.

External Examination.—Normally, the greater portion of the stomach (three-fourths) occupies the upper left quadrant of the abdomen, the remaining one-fourth lying to the right of the median lines. The cardiac orifice lies behind the sternal attachment of the sixth and seventh costal cartilages on the left side, while the pylorus is situated on the right side between the tip of the sternum and the junction of the seventh and eighth costal cartilages, and beneath the left lobe of the liver. In a moderately distended stomach, the highest part of the fundus is about the fifth interspace in the mamillary line, and the lowest border is one to two inches below the umbilicus in men, and one and a half to three inches below the same point in women. To determine these boundaries it is often necessary to inflate the stomach with air, after which the ordinary methods of physical diagnosis, inspection, palpation, percussion, and auscultation may be employed. *Traube's half-moon space* is an area on the left side of the trunk, bounded above by the upper edge of the sixth rib as far as the axillary line, on the right side by liver dullness, on the left side by splenic dullness, and below by the costal arch. When the stomach is empty and distended a tympanitic note is obtained over it by percussion, but when full, it yields a flat note. A pleural effusion on the left side may also render this area flat to percussion. *Leichtenstern's pulmono-hepatic angle* is the angle that exists at the junction of the lower edge of the left lobe of the liver, and the lower border of the left lung. Its apex lies behind the sixth rib, below the apex beat. The lung occupies it only during deep inspiration, and it is bisected by the pleural space. The angle is constantly maintained and filled in by the stomach.

Internal Examination.—To examine the stomach-contents it is necessary to administer a test-meal of definite quantity and quality, withdrawing it after a fixed time has elapsed. The digestive changes are then noted and deductions made as to the state of function of the stomach.

Baas and Ewald's test-meal consists of an ordinary roll weighing 9ʒ (35 Gm.) and 10 f℥ (300 Cc.) of water, or weak tea without milk or sugar. This is preferably given for breakfast, when the stomach is empty. It is removed at the end of one hour, and ordinarily from 20 to 40 Cc. should be the quantity withdrawn.

Leube-Riegel test-meal consists of beef-soup, 13-3 f℥ (400 Gm.), beefsteak, 6.6 ℥ (200 Gm.), bread, 1.6 ℥ (50 Gm.), and water 6.6 f℥ (200 Cc.). The contents of the stomach after this meal should be removed at the end of four hours.

In removing the stomach contents it is best to employ a soft, flexible, red rubber tube, closed at the inner end, but provided with lateral openings like a Nelaton's catheter, the length of which is about 3 feet (95 Cm.) The distance from the incisor teeth to the fundus is about 2 feet (60 to 65 Cm.), and the stomach tubes in common use are marked at this point on the tube. To introduce it, the tube should be moistened with water and passed well back into the pharynx, after which the patient is directed to swallow, and the tube is pushed on very gently. In withdrawing the contents it is best to employ siphonage, using a definite quantity of water so as to allow of its deduction in the chemical examination.

For the first thirty to forty-five minutes of gastric digestion lactic acid predominates, but at the end of an hour it is entirely replaced by free hydrochloric acid, which exists in quantities varying from 0.15 to 0.2 percent after a light meal to 0.2 to 0.33 percent after an abundant meal. To test for free acids it is common to employ filter paper which has been soaked in a solution of Congo-red. This turns blue in the presence of free acids. Tropeolin may be used in the same manner, paper soaked with it turning brown under similar circumstances. These do not differentiate, however, between the mineral acids and the organic acids.

Reaction of the stomach-contents is important from a clinical standpoint, and should, therefore, always be obtained. *Acidity* of the gastric contents from 0.15 to 0.2 percent is normal, and is due to hydrochloric acid. A greater percentage than this constitutes *hyperacidity* or *hyperchloridia* and is common in chlorosis, neurasthenia, hysteria, ulcer of the stomach, locomotor ataxia, etc. *Subacidity* refers to a deficiency in the mineral acid although acids of fermentation may be present. It is encountered in gastric cancer, neurasthenia, hysteria, gastric neuroses, anemia, chronic gastritis, etc.

hyperchlorhydria

Free hydrochloric acid may be ascertained by *Günzburg's, Boas'*, or *Tœpfer's test*. The reagent used by Günzburg consists of phloroglucin (2 Gm.), vanillin (1 Gm.), and absolute alcohol (30 Cc.). The gastric contents should be filtered and a few drops of this solution added to the filtrate. The mixture is evaporated to dryness, a beautiful rose-red tinge at the edge indicating free hydrochloric acid. This test is extremely delicate and will detect one part of hydrochloric acid in 20,000 parts of water. *Boas' reagent* is composed of resorcin (5 Gm.), white sugar (3 Gm.), and alcohol (100 Cc.). It is applied in the same manner as the preceding, the hydrochloric acid being indicated by a purple-red color. *Tœpfer's reagent* is a 0.5 percent alcoholic solution of dimethyl-amidoazobenzol. One or two drops added to 5 Cc. of the gastric filtrate will turn the mixture a bright cherry-red in the presence of free hydrochloric acid.

Vanill
Phlorogluc

To determine the total acidity of the gastric contents, the most convenient method is to add one drop of a 1 percent solution of phenolphthalein to 10 Cc. of the gastric filtrate and neutralize this mixture with a decinormal solution of sodium hydrate from a

Mohr's burette. A red coloration of the filtrate, which fails to disappear on shaking, indicates complete neutralization. The number of cubic centimeters of the sodium hydrate required is read from the scale and then multiplied by ten to obtain the percentage of total acidity, for instance, if 4 to 6 Cc. (the usual quantity) were employed for 10 Cc. of the filtrate, the percentage of total acidity would be 40 to 60. *The quantity of free hydrochloric acid* may easily be obtained (when it exists alone) if it is remembered that 1 Cc. of the alkaline solution is equivalent to 0.00365 Gm. of hydrochloric acid. Günzburg's reagent is applicable in this test as in the preceding for qualitative purposes. Tœpfer's reagent may also be employed in a similar manner for the same purpose. A one percent aqueous solution of alizarin is sometimes used; neutralization of the gastric filtrate containing a few drops of it, by the decinormal sodium hydrate solution is evidenced by a violet color. In all it is customary to employ 10 Cc. of the filtrate, using the reagents to test for the presence of the acid until it is completely neutralized by the alkali solution. (No increase of color)

X *Lactic acid* may be detected by Uffelman's or Arnold's test. *Uffelman's* reagent is composed of a 4 percent solution of carbolic acid, 10 Cc., distilled water, 20 Cc., and the official neutral ferric chlorid solution, one drop. The mixture should be freshly made and should possess an amethyst-blue color. The presence of lactic acid will be indicated by a canary-yellow color on mixing the reagent with the gastric filtrate. This test will detect one part of lactic acid in 20,000 parts solution. *Arnold's* reagent consists of two solutions: 1. A saturated alcoholic solution of gentian violet, 0.1 Cc. distilled water, 250 Cc.; 2. Official ferric chlorid, 5 Cc., distilled water, 20 Cc. In performing the test one Cc. of the first solution is mixed with one drop of the second in a test tube thereby producing a bluish-violet colored mixture. Contact with lactic acid changes it to a green or yellowish green color.

Butyric acid is distinguished by its characteristic odor on boiling the filtrate and by its brownish yellow reaction with Uffelman's solution. It may also be separated in drops by the addition of calcium chlorid.

Acetic acid may be recognized by its color or by the dark red color it produces with ferric chlorid solution. To obtain this, it is first extracted from the filtrate by ether, then evaporated, and the

x Kellings Test Best. page 249 Simon

residue dissolved in distilled water, after which it is neutralized by a solution of sodium carbonate.

Alcohol resulting from yeast fermentation in the stomach may be detected by Lieben's iodoform test. The gastric contents should be distilled, and a small quantity of liquor potassae added to the distillate. A few drops of a solution of iodin and potassium iodid (1, 2, 50) are then added, and if alcohol is present iodiform will be precipitated from the mixture.

Propeptone and peptone, the products of albumin digestion, yield a purplish red color on the addition of Fehling's solution.

Rennet is detected by its action on raw milk. A few drops of the gastric filtrate should be added to 10 Cc. of raw milk and the mixture placed in a chamber at an average temperature of 100° F. The presence of the ferment is indicated by coagulation of the milk. Rennet does not exist primarily as such, but as rennet-zymogen, which has no effect on milk. It may be converted into rennet by the addition of hydrochloric acid or calcium chlorid.

Pepsin is usually present in the filtrate, in which hydrochloric acid has been detected, but if it is absent it should be supplied in sufficient quantity and slices of coagulated white of egg added to the mixture. The whole is placed in an oven at 100° F., and in the presence of pepsin, dissolution of the eggs should occur within an hour.

Starch products in the filtrate will be indicated by the deep blue color produced by contact with iodin or Lugol's solution of iodin (1), potassium iodid (2), and distilled water (200). In the absence of such reaction it may be inferred that starch digestion has been complete.

The *rate of absorption* is indicated by the period necessary for free iodin to appear in the saliva after the ingestion of a capsule containing 0.1 Gm. of potassium iodid. Normally ten or fifteen minutes is sufficient. Filter paper, impregnated with starch, is used as the reagent.

The *motor power of the stomach* may be ascertained in three ways: 1. Withdrawal of the gastric contents six to seven hours after the ingestion of the Leube-Riegel test-meal should demonstrate no solid residue if the motor power is normal; 2. Salicyluric acid should appear in the urine within one hour after the ingestion of 1 Gm. of salol, under normal conditions. The acid is detected by the violet color produced on contact with a 10 percent solution of ferric chlorid; 3. The stomach is first washed out and 100 Cc. of olive oil are introduced through a tube. Two hours later the gastric contents are removed; if the motive power is not deranged it should be impossible to collect more than 20 to 40 Cc. of the oil.

Microscopic examination of the stomach-contents may reveal the presence of starch-cells, yeast-cells, muscle-fibers, shreds of mucous membrane, epithelium, Faden bacillus, Appler-Boas bacillus, pus-cells, blood corpuscles, and various bacteria.

ACUTE GASTRITIS.

Synonyms.—Acute simple gastritis; gastric fever; bilious fever; acute indigestion; subacute gastritis.

Definition.—An acute catarrhal inflammation of the mucous membrane of the stomach.

Causes.—It may arise from the presence in the stomach of undigested or indigestible foods, alcoholic beverages in excess, irritating medicines, such as the bromids, iodids, and arsenic, and corrosive poisons such as the mineral acids, corrosive sublimate, copper, carbolic acid, etc. It may also be due to exposure to cold and wet and the infectious fevers.

Pathologic Anatomy.—The mucous membrane of the stomach is swollen and engorged and covered with a grayish, semi-transparent tenacious mucus of alkaline reaction. Mucoid degeneration and cloudy swelling of the glandular cells are present. In *toxic cases,* erosions are observed everywhere throughout the mucosa; the gastric tubules are destroyed in great numbers; and the submucous, muscular, and serous coats may show decided destructive changes.

Symptoms.—Under ordinary circumstances, acute gastric catarrh is manifested by loss of appetite, heavily coated tongue, fetor of the breath, nausea, sometimes vomiting, first of undigested food, then viscid mucus, and finally bilious matter, moderate fever, headache, flashes of heat with sensations of burning in the palms of the hands and soles of the feet, abdominal pain, tenderness, and distress, eructations, vertigo, and constipation or diarrhea. Herpes may appear about the mouth towards the end of an attack. Jaundice may be present. The urine is scanty and contains lithates and pigment. Examination of the stomach-contents shows a deficiency in hydrochloric acid and an excess of lactic and fatty acids, mucus, and undigested food.

In *toxic gastritis,* immediately or very shortly after the poison is swallowed there ensues a deadly nausea, followed by rapid and persistent vomiting, first of the stomach-contents, afterwards shreds of mucous membrane and blood clots. Anxiety and depression, a weak, rapid pulse, slow and shallow respiration, cold, clammy skin, intense burning pain and heat in the epigastrium, thirst with burning in the fauces and gullet, exhaustive purging, shrunken features, and collapse follow.

Diagnosis.—Simple cases may resemble the onset of one of the infectious fevers, but the absence of other symptoms than those referable to the stomach, within 24 to 48 hours will establish the diagnosis. In toxic cases, the history and the sudden and severe symptoms will indicate poisoning, and the stains on the lips, mucous membrane, face, or clothing may determine the character of the poison, for instance: A blackish eschar points to *sulphuric acid,* a yellowish eschar to *nitric acid,* widespread softening and maceration of the tissues to *caustic potash,* whitish or glazed stains to *corrosive sublimate,* whitish and corrugated stains to *carbolic acid,* and yellowish white scars changing to grayish brown to *chromic acid.*

Prognosis.—In mild cases, the duration is about one week and the termination is favorable although complete recovery may be slow. The toxic form is very grave. Many perish from shock. In those not immediately fatal, death may occur from exhaustion and starvation incident to the destructive changes. Cases that eventually recover are always affected with gastric disturbances of varying degree.

Treatment.—The stomach should be placed at rest. When the stomach is overloaded ipecac should be given by the mouth or apomorphin hydrochlorate, gr. 1/8 (0.008 Gm.) should be administered hypodermically. If vomiting has already begun large draughts of hot water should be given. Active purgation by calomel, gr. v-x (0.32-0.65 Gm.) and sodium bicarbonate, gr. v (0.32 Gm.), followed by an ounce of magnesium sulphate or a full dose of Hunyadi Janos water, is of great value in most cases. Fractional doses of calomel are sometimes preferred. After the stomach and bowels have been thoroughly emptied, feeding should be resumed, beginning with the most bland food. Nux vomica, pepsin, or papoid may then be administered.

The following is an excellent stomachic sedative:

℞.	Sodii bicarb.	ʒ iij	12. Gm.
	Bismuth. subnit.	ʒ ij	8. Gm.
	Aq. chloroformi	f℥ iij	90. Cc.
M. et adde			
	Aq. menthæ pip.	f℥ j	30. Cc.
	Aq. lauro-cerasi	f℥ ij	60. Cc.

M. S.—Tablespoonful four times a day.

Another excellent formula after the acute symptoms have subsided is:

℞.	Strychninæ sulphat.	gr. ss	.03 Gm.
	Acid. hydrochlorici dil.	fʒ iv	15. Cc.
	Glycerini	f℥ j	30. Cc.
	Tinct. card. comp.	f℥ ss	15. Cc.
	Aq. lauro-cerasi	f℥ j	30. Cc.

M. S.—One teaspoonful, diluted, four times daily.

In *toxic gastritis*, morphin sulphate should be given hypodermically at once and repeated if necessary. Strychnin and atropin will be required in most cases to sustain the circulation. Demulcents and milk and lime-water should be freely given. Ice internally and locally affords great relief. Bismuth subnitrate, gr. xx-xxx (1.3-2 Gm.) every hour is beneficial. The stomach should be emptied of its contents immediately by means of the stomach tube or pump. If seen early the appropriate antidote should be administered. The following table will be found of great value for ready reference in this condition.

IRRITANT AND CORROSIVE POISONS.

Poison.	*Antidotes.*
Acids, mineral or oxalic	Chalk, magnesia (plaster of wall in emergency), solution carbonate soda, emollient drinks, fixed oils.
Ammonia ("household ammonia") or liniment.	Vinegar mixed with a bland oil, and sustain heart with strychnin.
Arsenic.	Moist peroxid of iron (obtained from perchlorid of iron and calcined magnesia) or the ferri oxidum hydratum, U. S. P., charcoal, ammonia to nostrils.
Carbolic acid.	Castor oil, olive oil, and the sulphate of magnesium or sodium as a chemical antidote. Prompt stimulation.
Chromic acid.	Acetate of iron, ten per cent. solution nitrate silver, forming insoluble silver chromate.
Corrosive sublimate.	Albumen, white of egg (four grains sublimate require white of one egg), flour, milk.
Iodin.	Demulcent drinks, starch or flour in water.
Phosphorus (matches).	Magnesia, turpentine, demulcents. Wash stomach by use of stomach tube or pump and warm water, afterward using Epsom salts.
Silver nitrate.	Solution common salt in demulcent drinks.
Soda, or "caustic potash." "Lye."	Olive oil, demulcents, vinegar, lemon juice, and hypodermically stimulants.
Sulphate copper.	Albumen or white of egg, stomach pump, and wash stomach with soap-water.

Alcoh
as Bran
whisky etc

CHRONIC GASTRIC CATARRH.

Synonyms.—Chronic gastritis; chronic dyspepsia; drunkards' dyspepsia.

Definition.—A chronic catarrhal inflammation of the stomach, with thickening of the coats and atrophy of the gastric glands.

Causes.—Repeated attacks of acute gastric catarrh; habitual and excessive use of spirituous liquors, tea, coffee, and the free use of ice-water during and between meals; improperly prepared and unsuitable food; irregularity of meals and imperfect mastication; excessive tobacco-chewing; malaria; disease of the heart, lungs, pleura, liver, or kidneys, producing chronic congestion of the stomachic vessels; cancerous or other degenerative diseases of the stomach.

Pathologic Anatomy.—The mucous membrane is of a brownish or slate color, elevated into ridges from hypertrophy, the result of constant congestion; the peptic glands first increase in size, then undergo granular change, resulting in atrophy of their cells. The mucous membrane is covered with a thick alkaline, tenacious mucus. Ewald describes the minute anatomy as that of a parenchymatous and interstitial inflammation, which may lead to such widespread degeneration of the glandular elements so that ultimately, scarcely a trace of secreting tissue remains. These changes may affect the entire organ or be limited to portions of the stomach. Van Valzah describes three forms of chronic gastritis—viz.: (1) Gastritis catarrhalis chronica, or chronic asthenic gastritis; (2) gastritis glandularis proliferans, or chronic hypersthenic gastritis; (3) gastritis glandularis atrophicans, or progressive atrophy of the gastric glands.

Symptoms.—The persistent and manifold symptoms of indigestion are present, such as loss of appetite, disagreeable feeling of gnawing and at times fullness in the stomach, tenderness in the epigastrium, but slightly influenced by eating, prominence of the epigastrium, from distention by decomposing gases, and occasional nausea and vomiting of undigested foods, after meals, or of colorless fluid when the stomach is empty.

Early morning vomiting of glairy mucus and saliva, coating of the tongue, constant thirst, burning at the pit of the stomach or under the sternum (heartburn), pain after eating, and constipation are common. In long-standing cases the circulation is feeble; there is depression of spirits often amounting to melancholia; and vertigo and sleeplessness are present. Follicular pharyngitis often accompanies the condition. The imperfect digestion leads eventually to loss of flesh. The urine is high-colored and contains phosphates, urates, and the oxalate of lime in excess. An examination of the gastric-contents will show a diminution in hydrochloric acid and ferments, and a large quantity of mucus.

Diagnosis.—Chronic gastritis may be readily recognized, if the history and symptoms are fully considered. In many instances the stomach disturbance is secondary to some visceral disease, which may be ascertained by a careful examination.

Gastric cancer may be distinguished by the early absence of hydrochloric acid, and the presence of large quantities of lactic acid and the Boas-Oppler bacillus in the stomach-contents, vomiting of a persistent character, containing blood in the advanced stages, enlargement at the pylorus, dilatation of the stomach, and cachexia.

Gastric ulcer differs from chronic gastritis in that there is hyperacidity, localized pain and tenderness, worse after eating, and hematemesis.

Prognosis.—With treatment, most of the symptoms may be greatly relieved and in mild cases cure may be effected. When the mucous membrane has become atrophied the outlook becomes unfavorable.

Treatment.—The first indication is the correction of the indigestion, which is usually the most pronounced and distressing symptom; this is accomplished by carefully regulating the amount and character of the food used, avoiding fatty, saccharine, and starchy articles or highly seasoned food or stimulants. A milk diet is beneficial, and to it may be added beef in small amounts, eggs, oysters, and a few fresh green vegetables. If beef is allowed, it had better, for a time, be in the form of "Salisbury steaks"—made of lean beef shaped into flattened cakes and broiled. This or whatever other articles of diet are allowed, should be taken an hour or more after sipping slowly a half pint (237 Cc.) of water, at 110°-150°. The hot water should also be taken before retiring.

The constipation should be relieved by the use of laxative mineral waters, such as Bedford, Saratoga, and Hunyadi Janos waters, or:

℞.	Magnesii sulph.	gr. lx-cxx	4.-8 Gm.
	Sodii et potass. tart.	gr. xxx-lx	2.-4. Gm.
	Acid. tartaric.	gr. xx	1.3 Gm.

M. S.—Dissolve in a glass of water and drink one hour before breakfast.

An excellent purgative and promoter of stomachic peristalsis is:

℞.	Ext. cascaræ sagradæ fld.	f℥ j	30. Cc.
	Glycerini	f℥ ss	15. Cc.
	Tinct. nucis vomicæ.	ff℥ ss	15. Cc.
	Aq. chloroformi vel inf. glycyrrh	f℥ j	30. Cc.

M. S.—One or two teaspoonfuls after meals, well diluted.

For the purpose of cleaning the stomach of the tenacious mucus as well as for its stimulating action on the glands, *lavage* or irrigation of the stomach with lukewarm water is valuable. The water may be medicated with a solution of salt, sodium bicarbonate, or boric acid. Ewald considers the morning, when the stomach is empty, the preferable time for lavage.

Those patients who object to lavage obtain relief from the systematic drinking of one-half to one pint (237-475 Cc.) of hot water an hour before meals, as mentioned above.

The irritable condition of the mucous membrane is at times greatly benefitted by the use of bromid of strontium, gr. xv (1 Gm.), well diluted, before meals. In the presence of some morbid condition of the mucous membrane the solution of the arsenite of potassium (Fowler's solution), ♏i-ii (0.06-0.12 Cc.) before meals, or bismuth subnitrate, gr. x-xx (0.65-1.3 Gm.), one hour before or two or three hours after meals will be of value. The following combination will be found very useful:

℞.	Sodii bicarb.	ʒ iv	15. Gm.
	Bismuth. subnit.	ʒ vj	24. Gm.
	Aquæ chloroformi	f℥ iij	90. Cc.
M. et adde			
	Aquæ lauro-cerasi	f℥ iij	90. Cc.
	Strychninæ sulph.	gr. j	.065 Gm.

M. S.—Two teaspoonfuls at meal-time in a little water.

Silver nitrate, gr. ¼ (0.016 Gm.), or silver oxid, gr. ss-i (0.032-0.065 Gm.) in pill, before meals, or dilute hydrochloric acid, ♏x-xv (0.6-1 Cc.) in water, before meals may also be employed. When pain is severe, opium, belladonna, or cocain may occasionally be required internally and belladonna plaster may be applied over the stomach.

To aid digestion, acids, pepsin, pancreatin, papoid, and bitters are of value, the following being an excellent prescription:

℞.	Pepsini (cryst.)	gr. lx	4.	Gm.
	Acid. hydrochlorici dil...	fʒ iv	15.	Cc.
	Glycerini	fʒ iv	15.	Cc.
	Strychninæ sulph.	gr. ss	.032	Gm.
	Aquæ chloroformi, q.s.ad	f℥ iij	ad 90.	Cc.

M. S.—One teaspoonful at meal-time in a little water.

In addition to medicinal measures, there should be prescribed mental and physical rest, gentle systematic exercises, change of environment, etc.

GASTRIC ULCER.

Synonyms.—Peptic ulcer; chronic gastric ulcer; perforating ulcer.

Definition.—A circumscribed solution of continuity, involving the mucous membrane and one or more layers, of which the walls of the stomach are composed; characterized by gastric pain, disorders of digestion, hyperacidity, and vomiting of blood.

Causes.—The important etiologic factors are early adult life, female sex, traumatism, chlorosis, and anemia. The exciting cause may be an embolus or thrombus, or self-digestion of the stomach wall. Similar ulcerations occur in the duodenum as the result of extensive superficial burns.

Pathologic Anatomy.—In the majority of cases the ulcer is solitary and is situated on the posterior wall near the pylorus. In a typical case there is a circular hole, with a sharp border in the mucous coat, the sides converging in the muscular coat, coming to a point in the serous coat, thus forming a funnel-shaped lesion. This appearance is most marked in recent cases. As the ulcer advances is becomes elliptical and irregular, varying from one-quarter to one-half inch in diameter. The edges, however, seldom become irregular. Perforation may occur. Blood-vessels are constantly eroded, producing profuse hemorrhage and subsequent hematemesis. Connective-tissue replaces the ulcerated area in the process of healing.

Symptoms.—Indigestion and its various manifestations are commonly present. The characteristic symptoms are pain, localized tenderness, vomiting, hematemesis, hyperacidity, and sometimes an enlargement in the region of the pylorus. The pain is paroxysmal in character and is aggravated by the taking of food; especially hot, cold, indigestible, or spicy substances. While usually located in the "pit" of the stomach it may radiate to the back and limbs. A burning, gnawing pain may also be present between the paroxysms of gastralgia. Tenderness on pressure is rather constant and usually may be elicited at a point an inch or two above the pylorus. Vomiting is common. It may appear immediately after eating, when the ulcer is near the cardiac orifice, but when located near the pylorus is usually comes on an hour after eating. The vomit consists of undigested food and mucus. Hemorrhage into the stomach from erosion of a blood-vessel causes vomiting of large quantities of bright red blood and the passage of dark blood from the bowels. It occurs in about 50 percent of cases. Examination of the stomach contents will show an increase in the hydrochloric acid. Anemia, loss of weight, anorexia, and general failing of health accompany the condition.

Diagnosis.—*Gastralgia* resembles gastric ulcer as regards hyperacidity and the paroxysmal pains. It differs in the absence of dyspeptic symptoms between the attacks, hematemesis, and localized tenderness and enlargement. Von Leube has shown that the application of an electric current during digestion causes a cessation of pain in gastralgia, but not in gastric ulcer and cancer.

Gastric cancer may be distinguished from gastric ulcer in that it occurs at a later period in life, has a more rapid course, the cachexia is more prominent, the pain is more constant, the growth is palpable, the vomit has a "coffee-grounds" appearance, and there is absence of hydrochloric acid and presence of lactic acid.

Intercostal neuritis accompanying chronic gastritis may lead to an error in diagnosis, but the absence of localized tenderness above the umbilicus, hyperacidity of the gastric contents, and hematemesis will serve to make a distinction.

Hyperchloridia is attended only by an increase in the hydrochloric acid and pain which is relieved by albuminous food. The other symptoms common to gastric ulcer are absent.

Chronic gastritis lacks hematemesis, the gastralic pains, and the localized tenderness.

Prognosis.—Usually the ulcer is slow in forming and runs a very chronic course, the average duration being about one year. Occasionally it may develop very suddenly. It may terminate in perforation (6 percent), peritonitis, fatal hemorrhage, or recovery with cicatricial formations. The mortality varies according to different observers from 10 to 30 percent. With proper treatment recoveries are frequent. Relapses are not uncommon occurrences.

Treatment.—The patient should be placed at rest in bed. The diet should consist of only the most bland food, preferably milk (one or two ounces every two hours) and lime-water, beef-juice, egg-albumin, or skimmed milk. When feeding by the mouth induces too much irritation, resort should be made to rectal alimentation. An enema made up of four ounces of milk, two eggs, a very small quantity of salt, and three drops of the tincture of opium, the entire mixture being predigested by pancreatin, is very valuable in this connection.

Severe hemorrhage will indicate the injection of warm normal salt solution into the rectum or hypodermically, and the administration of five drops of adrenalin solution (1 to 1,000). Strychnin, nitroglycerin, and ammonia may be given to sustain the heart at this period. Ergot, acetate of lead, gr. i-ii (0.065-0.2 Gm.), gelatin, tannic acid, gr. xv (1 Gm.), and persulphate or iron, gr. ¼-½ (0.0165-0.033 Gm.), are also advised to control the hemorrhage. An ice-bag over the stomach and small pellets of ice internally may be of value. Feeding by the mouth should be suspended when there is hematemesis.

The hyperacidity may be overcome by large doses of bicarbonate of soda and calcined magnesia every four hours. Bismuth subnitrate and belladonna may be useful. In the presence of constipation, Carlsbad salt should be employed. For relief of the pain, morphin may be necessary.

The medicines of special value in this condition are Fowler's solution, ♏i-ii (0.06-0.13 Cc.) every five hours, subnitrate of bismuth, gr. xx-xxx (1.3-2 Gm.), combined with sodium bicarbonate, gr. v (0.32 Gm.) three times daily, silver nitrate, gr. ¼-⅓ (0.016-0.022 Gm.) every four hours, and silver oxid, gr. ss. (0.032 Gm.). Iron and arsenical preparations are indicated for the constant anemia.

℞. Pulv. ferri albuminatis..gr. ij .13 Gm.
Sodii arseniat. ....gr. 1-20 .003 Gm.
M.—Ft. pil. or capsule.
S.—One three times daily.

The occurrence of perforation requires prompt surgical intervention.

GASTRIC CANCER.

Synonyms.—Cancer of the stomach; gastric carcinoma.

Definition.—A malignant growth, occurring for the most part at the pyloric extremity of the stomach, making constant progress, destroying the gastric tissues and infecting the lymphatic glands; characterized by disorders of digestion, pain, vomiting, marked anemia, and terminating in all cases by the death of the patient.

Causes.—Cancer of the stomach usually develops in women past forty years of age. Heredity is a factor in some cases and prolonged irritation such as accompanies the scars of old ulcers and chronic gastritis may be a predisposing cause.

Pathologic Anatomy.—The growth is nearly always primary. The most common variety is the scirrhus or hard cancer, next in frequency, the medullary or soft cancer, and the least frequent the colloid cancer. About 80 percent of gastric cancers are found at the pylorus. Those portions of the stomach remote from the growth are comparatively healthy. Scirrhus cancer gives rise to nodular growths and stenosis; medullary cancer is prone to ulcerate; and colloid cancer produces a diffuse infiltration and thickening of the stomach-walls. The growth usually begins in the tubules. The lymphatic glands adjacent to the stomach enlarge as the growth progresses and secondary cancers result.

Symptoms.—The manifestations of indigestion are present from the onset. The majority of cases have vomiting, occurring immediately after eating, if the disease is at the cardiac orifice, and some hours after, if located at the pylorus; if much dilatation of the stomach develops, the vomiting occurs some days after eating. The rejected matter is food in various stages of digestion, associated frequently with black grumous masses of altered blood and tissues. Hematemesis is frequent, rarely profuse, usually oozing of blood altered into a dark brown or black color—"coffee-ground" vomit or the oozing blood passes into the intestinal canal, causing tarry stools.

Absence of hydrochloric acid in the stomach is a very constant observation in gastric cancer. Boas and Stewart (D. D.), in 1895, found, by the use of the test-meal (flour soup), that lactic acid was always present in gastric cancer, and they were unable to find this acid in any other stomach condition. The Boas-Oppler bacillus is also present.

Pain is constant, but is dull and heavy in character, increased by pressure and food, and may radiate to the back. A tumor is found in three-fourths of the cases in the epigastric region which does not move with inspiration. Edema of the ankles is a common phenomenon in gastric cancer, occurring as early as the third month and may proceed to general anasarca. Marked anemia, emaciation, and cachexia are constant symptoms. Jaundice frequently occurs and the liver may be enlarged. There is involvement of the lymphatic glands in the supraclavicular and inguinal regions particularly. All the symptoms of gastric dilatation (q. v.) are present. The urine is albuminous and there may be irregular fever.

Diagnosis.—The age, sex, history, the presence of a palpable tumor, hematemesis of "coffee-ground" material, the absence of free hydrochloric acid and the presence of lactic acid and Boas-Oppler bacillus in the stomach-contents, and the cancerous cachexia are the important points in the diagnosis.

Chronic gastric catarrh is characterized only by aggravated dyspepsia and possesses none of the distinctive features of gastric cancer.

Gastric ulcer differs from gastric cancer in the age of the patient, the character of the pain, and the stomach-contents.

Abdominal tumors of other structures differ in that most of them in this region move on inspiration. Aneurysm of the abdominal aorta is distinguished by its expansile pulsation. A pulsation may be communicated to a scirrhus at the pylorus, but if the patient is directed to rest upon the hands and feet, the gastric tumor falls away from the aorta and the pulsation ceases.

Prognosis.—The disease usually lasts from one to two years, terminating in death by exhaustion, peritonitis, or hemorrhage. In persons under thirty years of age the disease is rapidly fatal. It may be complicated by fatty heart, thrombosis, or tuberculosis.

Treatment.—The treatment is unsatisfactory and is directed largely towards maintaining the patient's strength by suitable foods. Ordinary diet soon becomes inadequate and irritating, and predigested foods have to be used. Peptonized milk may be prepared by adding five grains of extract of pancreas and fifteen grains of sodium bicarbonate to a pint of milk, placing the mixture in a compartment at a temperature of 100° F., from which it is removed in one hour. Peptonized beef, peptonized eggs, and similar foods may be employed. Should the stomach become unretentive, or, in the presence of other contraindications, resort should be had to rectal alimentation. The administration of dilute hydrochloric acid aids digestion and prevents fermentation:

℞.	Strychninæ sulph. ......gr. ss	.032	Gm.
	Acid. hydrochlor. dil....fʒ iv	15.	Cc.
	Inf. condurango.q. s. ad f℥ viij	ad 240.	Cc.

M. S.—Tablespoonful before meals, diluted.

For *pain*, morphin, or the following recommended by Osler:

℞.	Morphinæ sulph. ....gr. ⅛	.008	Gm.
	Sodii bicarb. ....gr. v	.3	Gm.
	Bismuth. subnit. ....gr. x	.6	Gm.

M. S.—Repeated p. r. n.

Fetor of the breath may be relieved to some extent by carbolic acid, gr. ¼-⅓ (0.016-0.022 Gm.), or purified animal charcoal, gr. x-xxx (0.65-2 Gm.). Washing out of the stomach an hour before breakfast will produce benefit by removing the retained and fermented material in that viscus, but it is a dangerous procedure, and in the presence of ulceration, perforation may be produced. Stimulants should be avoided.

Surgical treatment has been of value in some cases.

GASTRIC DILATATION.

Synonyms.—Gastrectasis; pyloric obstruction: pyloric stenosis.

Definition.—An abnormal increase in the cavity of the stomach, with the walls either hypertrophied, or decreased in thickness; characterized by pronounced indigestion, vomiting of partly digested and partly decomposed food at intervals of a day or two, and noisy moving of flatus within the abdomen (borborygmus).

Causes.—Stenosis of the pylorus such as results from cancer, cicatricial contraction, hypertrophy of the pylorus, and the pressure of abdominal tumors is the most common cause. It may result from relaxation of the stomach-walls such as follows excessive eating or drinking, and anemia.

Pathologic Anatomy.—The entire organ is dilated and its muscular wall is hypertrophied in pyloric obstruction but in atonic dilatation, the muscular layer is thinner than normal, paler in color and presents signs of fatty degeneration. The mucous membrane is also pale, thin, and without rugæ.

Symptoms.—The characteristic feature of gastric dilatation is the vomiting which occurs long after meals, often at intervals of several days. The vomit is large in amount, and consists of undigested and fermented food and a turbid liquid. It contains yeast-cells and other low forms of plant life. In addition to vomiting, the symptoms of chronic gastritis and of the affection to which the dilatation is due are very prominent. Constipation is common.

Physical signs of gastric dilatation are: on *inspection,* abnormal prominence of the whole epigastric region, with a tumor in the pyloric region which seems to be connected with the stomach; *percussion,* if empty, tympanitic note having a metallic quality, extending to or below the umbilicus; if the stomach be filled, high-pitched bat note; *auscultation,* splashing and rumbling sound, the succussion sound being distinct if the body be shaken.

Diagnosis.—The peculiar vomiting and the physical signs together with the history will aid greatly in making a diagnosis. The outline of the stomach may be mapped out by physical examination by inflating the organ with air or filling it with liquid. The X-ray may also be used. A bismuth solution is given to produce a shadow in the stomach after which a skiagraph is taken.

According to Boas, dilatation is present when the greater curvature is below the umbilicus and when the greatest vertical diameter of the stomach is from 10 to 14 Cm. (4 to 5½ inches).

Prognosis.—Recovery is impossible in malignant pyloric obstruction, but in atonic dilatation considerable symptomatic relief may be afforded.

Treatment.—A "dry diet" should be used exclusively and only small quantities should be given at a time. Fluids should be administered by rectal enemas. Washing out of the stomach every night before retiring should be performed. An abdominal bandage may be of benefit. Drugs, such as dilute hydrochloric acid, nitromuriatic acid, pepsin, nux vomica, creasote, charcoal, salol, and bismuth betanaphtol may be employed to prevent fermentation. Surgical treatment, pyroplasty and gastroenterostomy, may be considered in organic pyloric obstruction.

GASTROPTOSIS.

Definition.—A displacement of the stomach downward, associated with prolapse of the bowel (enteroptosis or Glénard's disease) and often the prolapse of the kidney (nephroptosis).

Causes.—The condition is predisposed to by imperfect development of the abdominal and other muscles and by their early loss of tension with wasting. Women are most often affected. Frequent pregnancies, wearing of tight corsets or other unyielding garments, and occupations which favor stooping postures, such as sewing, tailoring, shoemaking, etc., are important factors in its production. Relaxation of the abdominal walls and loss of abdominal fat from any cause may give rise to displacement of the stomach.

Anatomic Conditions. — The transverse colon is the first organ to prolapse, and is soon followed by the ascending colon. The stomach is tilted, its lower border reaching below the umbilicus, while its lesser curvature lies between the ensiform cartilage and the umbilicus. In some cases the pyloric end is down to or below the umbilicus, without as much prolapse of the fundus. The right kidney is displaced and often floating or movable. The left kidney is less often displaced. Any or all of the conditions named may be associated with any of the organic gastric conditions.

Symptoms.—The patient complains of dyspepsia, abdominal distress and pain after eating, eructations of gases, anorexia, various nervous symptoms, weakness, and constipation.

Physical phenomena.—In the standing position the lower part of the abdomen projects and the upper part sinks in. In the recumbent position the abdomen shows a lateral extension. Aortic pulsation is frequent. There is often "a ridge lying across the abdomen" to be determined by palpation. Glénard termed this ridge the "cordecolique" transverse and thought it was due to a prolapse of and partial occlusion of the transverse colon. Other observers think it is the pancreas that is felt on account of the prolapse of the transverse colon. Inflation of the stomach often detects its prolapsed position with a lowered gastric splashing. The X-ray, with the aid of a bismuth subnitrate solution, will determine the location of the organ.

Treatment.—Abdominal bandages or some mechanical apparatus may be worn to help maintain the organ in place, but the benefit they produce is not marked. Surgical intervention often relieves the condition. In all cases, measures directed towards improving the general health should be employed. Lavage is useful in that it serves to prevent dilatation of the stomach.

GASTRIC HEMORRHAGE.

Synonyms.—Hematemesis; gastrorrhagia.

Causes.—Hemorrhage of the stomach may be due to ulcer, cancer, cirrhosis or congestion of the liver, scurvy, purpura, hemophilia, malaria, congestion of the spleen, chronic heart-disease, vicarious menstruation, traumatism, yellow fever, toxic gastritis, or rupture of an aneurysm into the stomach.

Symptoms.—The principal symptom is blood, of varying quantity, in the vomit. In ulcer of the stomach it is bright red, but in cancer, the most common cause, it has the characteristic "coffee-grounds" appearance, being dark, mixed with the food, and of acid reaction. If the hemorrhage is profuse, blood will appear in the stools. There are also present at the time of the loss of blood pallor, weakness, ringing in the ears, faintness, and a sinking feeling at the pit of the stomach.

Diagnosis.—*Hemoptysis*, hemorrhage from the lungs, is distinguished from hematemesis in that the blood is coughed up and is bright red, frothy, and of alkaline reaction. Physical examination of the chest will serve to detect the origin in some pulmonary or cardiac disease.

Prognosis.—Except in case of a ruptured aneurysm, hematemesis is seldom the direct cause of death. Hemorrhage from the stomach in the course of gastric ulcer or cancer, hepatic cirrhosis, hemophilia, and the infectious fevers is an unfavorable sign. The outcome depends entirely on the underlying cause.

Treatment.—Rest in bed is absolutely necessary and food should be temporarily withheld. Pellets of ice may be swallowed and ice-bags should be placed over the stomach and along the spine. In some cases hot water is equally beneficial. Morphin and ergotin should be given hypodermically. Monsel's solution, ♏i-v (0.06-0.3 Cc.), diluted, or adrenalin (1 to 1000), ♏x (0.6 Cc.), may be administered by the mouth for their hemostatic effect. Tannic acid, lead acetate, and gelatin may also be employed for the same purpose. Shock should be treated as under ordinary circumstances. The condition underlying the gastric hemorrhage should receive attention.

GASTRALGIA.

Synonyms.—Cardialga; gastrodynia; stomachic colic; spasm of stomach; neuralgia of the stomach.

Definition.—A painful condition of the stomach, induced by various forms of irritation; characterized by violent paroxysms of gastric pain and associated with feeble cardiac action, and symptoms of collapse but independent of disturbance of the gastric functions.

Causes.—The affection belongs to the group of neuralgias. The most important factor in its causation is general nervous depression or neurasthenia; other causes are gastric cancer or ulcer, malaria, rheumatic or gouty diathesis, syphilis, anemia, and certain articles of diet. It occurs in chronic nervous affections as the so-called "gastric crises." It is more frequently observed in women than men, and may arise from worry, menstrual disorders, sexual excesses, and the abuse of tobacco.

Symptoms.—Romberg's description of an attack may be quoted: "Suddenly, or after a feeling of pressure at the precordium, there is severe griping pain in the stomach, usually extending to the back, with a feeling of fainting, a shrunken countenance, cold hands and feet, and an intermittent pulse. The pain becomes so excessive that the patient cries out. The epigastrium is either puffed out, like a ball, or retracted, with tension of the abdominal walls. There is often pulsation in the epigastrium. External pressure is well borne, and not unfrequently the patient presses the pit of the stomach against some firm substance, or compresses it with his hands. Sympathetic pains often occur in the thorax, under the sternum, and in the esophageal branches of the pneumogastric, while they are rare in exterior of the body. The attack lasts from a few minutes to half an hour or longer; then the pain gradually subsides, leaving the patient much exhausted; or else it ceases suddenly, with eructation of gas or watery fluid, or with vomiting, and with a gentle, soft perspiration, or with the passage of reddish urine."

Diagnosis.—*Myalgia of the abdominal muscles* is distinguished by tenderness on pressure over the affected area, more constant pain and the absence of symptoms directly referable to the stomach.

Gastric cancer is differentiated by the age, course, history, hematemesis, cachexia, tumor, and the constant character of the pain.

Gastric ulcer is attended by localized pain and tenderness, aggravated by food and external pressure, hematemesis, hyperacidity, and dyspeptic symptoms.

In *biliary colic* the pain is usually to the right of the median line, radiating to the right and to the right scapula and shooting towards the right ilium. Chills, fever, and jaundice are also present.

In *renal colic* the pain begins at the kidney and radiates along the corresponding ureter. The pain is mostly posterior.

Abdominal colic is attended by gaseous distention and is centered lower down in the abdomen.

Angina pectoris is characterized by pain, which radiates from the heart down the left arm and accompanied by a sense of constriction of the thorax, and a strong fear of impending death.

The gastric crisis of locomotor ataxia may be recognized by the concomitant signs, the characteristic gait and pupils, history, etc.

Prognosis.—The affection is not dangerous to life but may persist for an indefinite period.

Treatment.—During the paroxysm chlorodyne mx-xxx (0.6-2 Cc.), may be administered for the relief of the pain, as the occasion requires. A mild attack may be relieved by antipyrin, gr. x (0.65 Gm.), and the application of a hot-water bag over the stomach. Galvanism, placing the anode over the stomach and the cathode near the spine, is often beneficial. When the pain is very severe morphin, by hypodermic injection, may be necessary. In recurring attacks Van Valzah recommends:

℞.	Codein	gr. ¼	.016 Gm.
	Ext. cannab. indicæ	gr. 1-10	.006 Gm.
	Atropinæ sulphas	gr. 1-200	.00032 Gm.
	Aconitinæ	gr. 1-400	.00016 Gm.

M.—Ft. capsul.
S.—One every four or six hours.

During the interval, the underlying cause should be ascertained and appropriate treatment instituted. As nerve-exhaustion is usually the cause, rest, regulated diet, exercise of moderate degree, fresh air, nerve-tonics, etc., should be prescribed.

ATONIC DYSPEPSIA.

Synonyms.—Dyspepsia; indigestion; heartburn; pyrosis.

Definition.—A functional derangement of the stomach, with either deficient secretion in the quantity or quality of the gastric juice; characterized by disorders of the functions of digestion and assimilation and the presence of various nervous symptoms.

Causes.—Among the principal etiologic factors may be mentioned nervous depression from worry and fatigue, sedentary habits, imperfect mastication, ingestion of large quantities of food, unchanged diet, heredity, neurasthenia, hysteria, and the female sex.

Symptoms.—The appetite is capricious, perverted, or lost; digestion is difficult, there is a sense of distention and weight in the epigastrium; and there is acidity of the gastric contents from decomposition of the albuminoids. Heartburn, flatulency, regurgitation of portions of partly-digested food or acrid fluid-water-brash or pyrosis, and pain or soreness at the pit of the stomach during digestion are also present. There is drowsiness after meals and insomnia at night. Defective memory, headache, absent mental vigor, flashes of heat, followed by more or less perspiration, and palpitation may be manifested. The tongue is usually broad, flabby, and pale and shows marks of the teeth. The bowels are constipated and the urine is scanty, high-colored, and contains an excess of urates and oxalates; in the nervous type, it is pale, of low specific gravity, and contains phospates.

Varieties of Dyspepsia.—I. *Nervous dyspepsia,* atonic form, seen in active business or busy professional men, especially those of thin, spare build, of nervous temperament, who eat rapidly and hurry off immediately to their business. These cases present all the marked nervous phenomena, such as drowsiness after meals, and feeling worse after a nap; inability for mental exertion after meals, defective memory, headaches, and at times vertigo and sleepless nights. II. *Flatulent dyspepsia,* seen in hysterical individuals, and showing immense development of gas throughout the abdomen, associated with vertigo and mental worry or hypochondria. III. *Acid dyspepsia,* water-brash, seen when the diet is coarse, characterized by acidity of the gastrointestinal canal and of the urine. IV. *Irritative dyspepsia.* Vomiting a prominent symptom. In these cases the tongue is small, red, and pointed.

Prognosis.—With the institution of proper treatment the outlook is favorable, otherwise the duration is indefinite.

Treatment.—Regulation of the diet is of great importance. Saccharine, starchy, or fatty articles of food should be interdicted. Mastication should be slow and complete and only small quantities of food should be taken at a time. Underdone meats, "Salisbury steaks," eggs, fish, oysters, and green vegetables with stale or brown bread are advised. Stimulants should not be taken with the meals, and only small quantities of liquids should be allowed. Rest of a half to an hour's duration after meals is of benefit. General physical and mental rest is indicated in the nervous type.

The medicinal treatment embraces a great number of remedies. As aids to digestion, taka-diastase, gr. ii-iv (0.13-0.26 Gm.), or the following formulas may be prescribed:

℞. Pepsini puræ ....ʒ j	4. Gm.
Acid. hydrochlorici dil..fʒ iv	15. Cc.
Glycerini ....fʒ iv	15. Cc.
Aq. lauro-cerasi ....f℥ ij	60. Cc.

M. S.—One teaspoonful with meals, diluted.

℞. Papoid (puræ) ....gr. xxx	2. Gm.
Sodii bicarb. ....gr. lx	4. Gm.
Pulv. zingib. jam........gr. v	.3 Gm.

M.—Ft. capsul. or pil. No. xx
S.—One at meal-time and bed-time.

To stimulate peristalsis, nux vomica, gentian, or cinchona may be used, and for the acidity, alkalies, particularly bicarbonate of soda, may be given. Purified animal charcoal, gr. x-xx (0.65-1.3 Gm.) or one of the carminatives will relieve flatulency. Pyrosis may be benefitted by the administration of bismuth subnitrate, gr. xx (1.3 Gm.), and aromatic powder, gr. v (0.32 Gm.). Vomiting may be overcome by the use of sodium or strontium bromid, gr. v (0.3 Gm.), carbolic acid, gr. ⅙-¼ (0.011-0.016 Gm.), or chloral hydrate, gr. x-xv (0.65-1 Gm.). Irrigation of the stomach, or the drinking of one-half to one pint of hot water an hour before meals is very beneficial. In anemic cases, iron, quinin, strychnin, and arsenic will be required. For constipation Hunyadi water, resin of podophyllum, or the following may be used:

℞. Sodii bicarbonatis ......ʒ ij	8. Gm.
Tinct. nucis vomicæ....fʒ iv	15. Cc.
Tinct. capsici ....fʒ j	4. Cc.
Tinct. rhei ....f℥ iss	45. Cc.
Inf. gentian. comp....ad f℥ vj	ad 180. Cc.

M. S.—Half tablespoonful after meals, in water.

℞. Ext. cascaræ sagradæ fld. f℥ j	30. Cc.
Tinct. nucis vomicæ.....f℥ ss	15. Cc.
Syr. zingib..............f℥ ss	15. Cc.
Inf. sarsaparillæ..q. s. ad f℥ iij	ad 90. Cc.

M. S.—Teaspoonful three times daily, diluted.

DISEASES OF THE INTESTINES.

INTESTINAL INDIGESTION.

Intestinal indigestion or dyspepsia is a functional derangement due to defects in the various intestinal secretions, or deficient peristalsis, or both resulting in more or less complete decomposition of the chyme; characterized by abdominal pain and distention, tympanites developing some hours after meals, emaciation, anemia, and various nervous symptoms.

Causes.—It may be inherited or it may be due to imperfect diet, over-eating, irregular meals, deficient exercise, worry and mental fatigue, immoderate use of tobacco, or stimulants, diseases of the stomach, intestinal tract, liver, or pancreas, or malaria.

Symptoms.—The affection may be acute or chronic.

The acute form is usually the result of an irritant in the duodenum and is attended by rapidly developed pain, flatulency, borborygmi, slight fever, coated tongue, loss of appetite, headache, and diarrhea. In sudden attacks, the accumulated gases often cause paroxysms of colic. Severe attacks are associated with jaundice, light-colored stools, and high-colored urine, indicating hepatic disturbance. In such cases, the onset is accompanied by malaise, chilliness, fever, 100°-102° F., increased pulse, headache with or without vomiting, coated tongue, abdominal pains increased on pressure, tympanites, cramps in the legs, and diarrhea. The stools are at first soft and normal with fecal odor and color becoming later frothy, watery, of a peculiar odor, and made up of mucus and undigested food. Their reaction is alkaline, and the microscope shows epithelial, round, and blood-cells, Charcot's crystals, crystals of the oxalate of calcium, calcium phosphate, etc.

The chronic variety follows varying grades of decomposition in the pasty, digested food after it has left the stomach. It is attended by pain, two to four to six hours after meals, with tenderness and distention in the upper abdomen, tympanites, borborygmi, dyspnea, and constipation. Anemia, emaciation, functional derangement of the liver, and marked nervous phenomena develop as the affection progresses. The skin is harsh and dry, and the urine is high-colored, of increased specific gravity, and acid in reaction, and on cooling deposits lithates, uric acid, and oxalate of lime crystals.

Diagnosis.—The late appearance of the symptoms after ingestion of the meals is the main feature in distinguishing intestinal indigestion from gastric indigestion. Usually they exist more or less combined.

Treatment.—In the *acute variety*, opium should be given and heat applied to the abdomen to relieve the distress, and a cathartic, preferably calomel, followed by a saline, administered to expel the irritant.

℞.	Hydrarg. chlor. mit.	gr. ⅓	.02 Gm.
	Sodii bicarb.	gr. ij	.13 Gm.
	Pulv. ipecac.	gr. ⅙	.01 Gm.
	Sacch. lact.	gr. iij	.2 Gm.

M.—Ft. charta.

S.—One every 2 hours until six have been taken.

After which stimulate the gastro-intestinal canal with:

℞.	Tinct. nucis vomicæ.	fʒ iv	15. Cc.
	Acid. hydrochlorici dil.	fʒ iv	15. Cc.
	Tinct. card. comp.	fʒ iv	15. Cc.
	Ess. pepsin.	q. s. ad f℥ iij	ad 90. Cc.

M. S.—Teaspoonful every three hours, diluted.

For the more severe variety of intestinal indigestion (or catarrh), wash out large bowel with—

℞.	Magnesii sulph.	℥ j	30. Gm.
	Glycerini	f℥ j	30. Cc.
	Aquæ bul.	f℥ iv	120. Cc.

M. S.—Slowly inject into bowel from a fountain syringe.

Internally either of the following excellent combinations:

℞.	Naphthalini	gr. xxx	2. Gm.
	Bismuth. salicyl.	gr. lxxx	6. Gm.
	Acid. carbolici	gr. iv	.26 Gm.
	Glycerini	f℥ j	30. Cc.
	Aq. chloroformi	f℥ iij	90. Cc.

M. S.—Two teaspoonfuls every two or three hours, diluted.

Or—

℞.	Sodii phosphat.	℥ j	30. Gm.
	Acid. phosph. dil.	fʒ iv	15. Cc.
	Syr. limonis	f℥ j	30. Cc.
	Aq. chloroformi	f℥ iij	90. Cc.
	Aq. menth. pip.	f℥ iiss	100. Cc.

M. S.—One tablespoonful after meals, well diluted.

Chronic cases require the administration of laxatives such as Bedford, Friedrichshall, Pullna, or Hunyadi Jânos waters, resin of podophyllum, or fluid extract of cascara sagrada and intestinal digestants. Pancreatic liquor fʒ.i-iv (4-15 Cc.) or pancreatic extract, gr. ii-v (0.13-0.3 Gm.), with bicarbonate of soda, gr. v-x (0.3-0.6 Gm.), two or three hours after meals is of great benefit. Purified oxgall, gr. i-iii (0.065-0.2 Gm.) after meals or the following may also be employed:

℞.	Papoid	gr. j-ij	.065-.13 Gm.
	Naphtalini	gr. j	.065 Gm.
	Ext. nucis vomicæ	gr. ⅓	.022 Gm.

M.—Ft. pil.

S.—One such to be taken every four or six hours.

Excellent results follow the use of the following pill:

℞.	Sodii arseniat	gr. 1-20	.003 Gm.
	Strychninæ sulph.	gr. 1-32	.002 Gm.
	Pepsinæ puræ	gr. ij	.13 Gm.

M. S.—After each meal.

The diet should be restricted in amount and confined almost entirely to articles which are readily digested in the stomach, such as beef, eggs, and milk.

INTESTINAL COLIC.

Synonyms.—Enteralgia; tormina; gripes.

Definition.—A spasmodic contraction of the muscular layer of the intestinal tube; characterized by acute paroxysmal pain near the umbilicus, relieved by pressure, and associated with feeble cardiac action.

Causes.—Intestinal colic may be due to constipation, the presence of indigestible food, or an abnormal quantity of bile in the intestinal tract, structural lesions of the intestinal wall, lead-poisoning, syphilis, gout, rheumatism, locomotor ataxia, malaria, hysteria, or reflex causes.

Symptoms.—Paroxysmal pain of a tearing, cutting, pressing, twisting, pinching, or bearing-down character centering around the umbilicus is the most prominent symptom. The abdomen is tense and pressure upon it relieves the pain. In severe attacks, the surface is cold; the features are pinched; the pulse is small and hard, and there may be nausea, vomiting, and tenesmus. Constipation is usually present. The duration is from a few minutes to several hours, often with intermissions. A discharge of flatus is the usual termination.

Diagnosis.—*Gastralgia* differs from colic, in the pain being in the epigastric region and associated with disorders of digestion.

In *hepatic colic*, or the pain due to the passage of gallstones, the pain is in the hepatic region, attended with soreness over the gall-bladder, and retching and vomiting, followed by jaundice and the presence of bile in the urine.

In *nephritic colic*, the pain follows the course of one or both ureters, shooting to loins and thigh, with retraction of the testicle of the affected side, strangury, and bloody urine.

In *uterine colic*, the pain is in the pelvis, and associated with menstrual disorders, in fact, a dysmenorrhea.

In *ovarian colic* or neuralgia, there is pain on pressure over the ovaries, with hysteric phenomena.

Inflammatory disorders of the abdomen differ from colic by the presence of fever and tenderness on pressure.

Lead colic is always preceded by symptoms of lead-poisoning, to wit: slate-colored skin, dark gums showing a blue line, heavy breath, with sweetish metallic taste, obstinate constipation, impaired appetite, slow pulse, and contracted abdominal walls.

Appendicitis may be distinguished by the localized pain and tenderness in the right iliac region, induration, and muscular rigidity.

Prognosis.—Favorable.

Treatment.—The pain should be relieved by turpentine stupes over the abdomen, carminatives, and the hypodermic injection of morphin sulphate, gr. 1/6-1/3 (0.011-0.22 Gm.). In all cases, blue mass, gr. v-x (0.3-0.6 Gm.) or calomel, gr. 1/2 (0.03 Gm.) every half hour until four of five grains have been taken, should be administered and followed by a saline cathartic. In the interval, the cause should be ascertained and removed.

In lead colic, morphin, castor oil, or sulphate of magnesia, potassium iodid, syrup of hydriodic acid, and olive oil are indicated.

CONSTIPATION.

Synonyms.—Intestinal torpor; costiveness.

Definition.—A functional inactivity of the intestinal canal, either due to atony of the muscular coat, causing lessened peristalsis, or to deficiency of intestinal and biliary secretion; characterized by a change in the character, frequency, and quantity of the stools.

Causes.—Diseases of the digestive tract, fevers, diseases that lessen intestinal secretions, affections that diminish peristalsis, sedentary habits, neglect, improper food, malaria, lead-poisoning, atony of the intestinal and abdominal walls, strictures, displaced organs, and foreign bodies are the common causes.

Symptoms.—One stool in twenty-four hours may be taken as an indication of the normal state as regards the intestinal tract; less than this constitutes constipation, although it may be unattended with any discomfort, for a considerable period. The change in number and consistency gives rise eventually to straining, distress, tenesmus, and irritation of the rectum. These are followed by dyspeptic symptoms, headache, mental torpor, vertigo, palpitation, and often abdominal distention.

Prognosis.—The outlook is favorable but the course is likely to be indefinite. Hemorrhoids, impaction, anal fissure, ulceration, and similar conditions may occur as sequels.

Treatment.—In all cases a careful examination should be made to ascertain the cause, which should be promptly removed. A large portion of the treatment in ordinary cases rests with the patient.

First, the patient must have a regular hour each day for going to stool, and must remain a sufficient time to permit a thorough evacuation of the bowels, assisting, until habit of daily stools is formed, by a warm water injection.

Second, the diet must be carefully regulated, as concentrated foods increase the costive habit, so that those predisposed should eat bulky foods, much vegetables and fruits. Bran bread, gluten bread, water-drinking, cornmeal, and oatmeal should be advised.

Third, purgative mineral waters, such as Saratoga, Bedford, Apenta, Carlsbad, Friedrichshalle, and Hunyadi Janos should be cautiously employed. Epsom salt or Rochelle salt is a very efficient drug in this condition. Aloin, gr. ⅛-¼ (0.008-0.016 Gm.), after meals, or glycerin in enema or suppository may also be employed to combat the constipation. The tone of the intestinal wall may be restored by electricity or kneading of the abdominal walls, systematic exercise, cold bathing, and massage.

Fourth, the administration of one of the following formulas:

℞.	Ext. nucis vomicæ	gr. ¼	.016 Gm.
	Ext. belladonnæ (alco.)	gr. ¼	.016 Gm.
	Ext. aloes (aqua)	gr. ss	.032 Gm.
	Pulv. rhei	gr. j	.065 Gm.
	Olei cajuputi	♏ j	.06 Cc.

M. S.—In pill, at bedtime; and after a week, every second or third night.

℞.	Resina podophyl.,		
	Ext. physostig.,		
	Ext. belladonnæ (alco.),		
	Aloïni	āāgr. ¼	āā .016 Gm.

M. S.—In pill, every night, or second or third night.

℞.	Ext. cascaræ sagradæ fld.	♏xx	1.3 Cc.
	Glycerini	♏xx	1.3 Cc.
	Syr. sarsaparillæ	♏xx	1.3 Cc.

M. S.—To be taken one hour after meals, or once a day, as indicated.

DIARRHEA.

Synonyms.—Enterorrhea; alvine flux; purging.

Definition.—Frequent loose alvine evacuations, without tenesmus; due to functional or organic derangement of the small intestines, produced by causes acting either locally or constitutionally.

Causes.—Among the local causes, may be mentioned indigestion, indigestible food, impure food and water, irritating matters or secretions poured into the bowels, intestinal inflammation, and entozoa. The general causes include atmospheric changes, sudden mental shock, purgatives, certain infectious fevers, and cachectic conditions such as attend tuberculosis, pyemia, Bright's disease, cancer, diabetes, etc.

Symptoms.—Diarrhea may be acute or chronic and is manifested chiefly by an alteration in the number and character of the stools. *Mucous stools* are those in which there are great quantities of mucus, indicating inflammation of the lower bowel. *Lienteric stools* contain considerable undigested food and point to inflammation of the stomach and upper bowel. *Watery or serous stools* occur in nervous and colliquative diarrheas, enteritis, cholera, and similar affections. *Green stools* may be due to an excess of bile, bacterial growth, or marked alkalinity of the digestive tract. *Fatty stools* are produced by the ingestion of large quantities of fatty foods, pancreatic diseases, and the absence of bile. *Purulent stools* arise from ulceration along the intestinal canal or the rupture of adjacent abscesses into the bowel. *Black stools* may be due to the presence of blood from hemorrhages high up in digestive tract, bismuth, charcoal, tannate of iron, etc. *Red stools* may result from the presence of fresh blood or the administration of diarrhea mixtures containing hematoxylon. *Bloody stools* or melena follow hemorrhage from any portion of the digestive tract and result from inflammation, ulceration, traumatism, infectious fevers, chronic heart, liver, or kidney disease, infarction, hemorrhoids, anal fissure and fistula, rupture of an aneurysm, scurvy, purpura, and vicarious menstruation.

Acute diarrhea presents itself in several forms. In the feculent form which results from indiscretions in diet, intestinal parasites, and indigestion, the patient experiences within a few hours after meals colicky pains, nausea, flatulency, and a desire for stool. The tongue may be coated. Purging relieves the pain. The stools are composed of a brown fluid and feces, and are very offensive. Their color becomes lighter after four or five evacuations. The duration is seldom more than two or three days. In the lienteric variety, the food passes through unaltered or very slightly digested. The stools are frequent and in addition to the undigested food, there are bile, mucus, and serum. Emaciation is common. In the bilious form, which is due to excess of bile, griping pains in the abdomen and scalding sensations at the anus are present, and the stools are green or yellow.

Chronic diarrhea results from the persistence of acute diarrhea or constitutional affections. The stools continue frequent, but are paler in color. Emaciation, anemia, dyspepsia, etc., accompany this affection.

Prognosis.—As diarrhea is only a symptom its prognosis depends upon the underlying condition. In the feculent and bilious forms it is favorable, but in the lienteric and chronic forms, when emaciation begins, it assumes an unfavorable character.

Treatment.—*Acute diarrhea.* If the tongue is heavily coated, the breath fetid, and the stools not excessive in number, it is well to clear the intestinal canal with a laxative such as castor oil or a saline. For children between one and two years of age:

℞.	Pulv. ipecac.	gr. ss	.032 Gm.
	Pulv. rhei	gr. ¼-⅓	.016-.022 Gm.
	Sodii bicarb.	gr. ss-ij	.032-.13 Gm.

M. S.—Every four hours until the character of the stools changes.

As a rule, however, the stools have become so frequent when advice is sought that the time for laxatives has passed, and some one of the following combinations is indicated:

℞.	Salol	gr. xx-xxx	1.3-2. Gm.
	Bismuth subnit.	ʒ j	4. Gm.
	Sacch. lac.	ʒ j	4. Gm.

M.—Ft. chart. No. x.

S.—One every two or three hours, reducing the dose for children.

Or—

℞.	Bismuthi salicylat.	gr. xxx	2. Gm.
	Morphinæ sulph.	gr. j	.065 Gm.

M.—Ft. chart. No. vj.

S.—One every three hours.

Or the following modification of "Squibb's diarrhea mixture":

℞.	Tinct. opii deodorat....	fʒ iv	15. Cc.
	Tinct. camphoræ	fʒ iv	15. Cc.
	Tinct. capsici	fʒ ij	8. Cc.
	Chloroformi puræ	fʒ iss	6. Cc.
	Spts. vini gallici.......	f℥ j	30. Cc.
	Vini pepsini	ad f℥ iij	ad 90. Cc.

M. S.—One teaspoonful, p. r. n.

Or the following, which I have always found successful:

℞.	Tinct. opii deodorat....fʒ iv	15. Cc.
	Spts. chloroformi ......fʒ ij	8. Cc.
	Acid. sulphuric. dil.....f℥ j	30. Cc.
	Vini pepsini ...q. s. ad f℥ iij	ad 90. Cc.

M. S.—One teaspoonful in water after each stool.

For the *bilious* form:

℞.	Hydrargyri chlor. mitis..gr. ⅛	.008 Gm.
	Sodii bicarb. ....gr. ij	.13 Gm.
	Pulv. opii ....gr. ¼	.016 Gm.

M. S.—Every two or three hours until eight powders are used, followed by large doses of bismuth and pepsin.

In all acute forms, restricted and regulated diet are imperative, pure milk with lime-water being the most suitable.

In adults, an opium suppository often checks a flux that is uninfluenced by opium internally.

In *lienteric* or dyspeptic diarrhea a carefully regulated diet and either of the following combinations:

℞.	Pepsini glycerit. ....f℥ j	30. Cc.
	Liq. potassii arsenit.....♏xx	1.3 Cc.
	Tinct. opii deodorat....fʒ ij	8. Cc.
	Aq. chloroformi..q. s. ad f℥ iij	ad 90. Cc.

M. S.—One teaspoonful at meal-time.

Or—

℞.	Papoid ....gr. xx	1.3 Gm.
	Bismuth. subnit. ....ʒ j	4. Gm.

M.—Ft. chart. No. x.

S.—One at meal-time.

Chronic diarrhea. Bismuth, gr. xxx-xl (2-2.6 Gm.), in milk, every four hours; Hope's camphor mixture, f℥j (30 Cc.), every four hours; or copper sulphate, gr. 1-12 (0.005 Gm.), extract of opium, gr. 1-12 (0.005 Gm.), every four hours; or silver nitrate, gr. ⅙ (0.01 Gm.), extract of opium, gr. ⅕ (0.011 Gm.), every five hours; may all be used with more or less success; when dry tongue and great flatulency are present use:

℞.	Ol. terebinthinæ ....fʒ j	4. Cc.
	Ol. amygdal. express...f℥ ss	15. Cc.
	Tinct. opii ....fʒ ij	8. Cc.
	Mucil. acaciæ ....fʒ iv	15. Cc.
	Aq. lauro-cerasi ....f℥ ss	15. Cc.

M. S.—One teaspoonful every three or four hours, diluted.

The diet should be nutritious in character, and moderate stimulants are indicated. Activity of the skin and kidneys should be encouraged.

All varieties of intestinal catarrh or diarrhea are benefitted by a few days' rest in bed and daily hot baths.

CATARRHAL ENTERITIS.

Synonyms.—Intestinal catarrh; acute diarrhea; inflammation of the bowels.

Definition.—A catarrhal inflammation of the mucous membrane of the small intestines; characterized by fever, pain, tenderness, and looseness of the bowels. When the catarrh is limited to the duodenum it is termed *duodenitis,* and is attended by slight jaundice.

Pathologic Anatomy.—There first ensues hyperemia of the mucous membrane and intestinal glands, manifested by redness, swelling, and edema; this is followed by increased secretion, and an overgrowth and desquamation of the epithelium, together with a copious generation of young cells. As a result of the hyperemia, rupture of the capillaries and extravasation of blood often occur.

The swollen glands show a strong tendency to ulcerate. This catarrhal process may involve the entire tube or be limited to portions of it. If the catarrhal changes extend to the ileum, the solitary and Peyerian glands show swellings that might be mistaken for the changes of typhoid fever.

Causes.—In some cases, a specific virus seems to be the etiologic factor. Ptomain poisoning, such as follows the ingestion of decomposed food and milk, improper and indigestible food, summer season, exposure to cold and wet, while perspiring, irritants such as foreign bodies in the intestinal tract, childhood, imperfect hygiene, and inorganic poisons may be mentioned as the most common causes.

Symptoms.—The *acute form* begins with languor, chilliness, fever 102°-103° F., anorexia, colicky pains, and localized abdominal tenderness. Nausea and vomiting often occur. The bowels are at first constipated, but later diarrhea supervenes. The stools contain but little fecal matter; are yellow or greenish yellow in color, and mixed with undigested food. When very numerous they become thin and watery, constitiutng the so-called "rice-water" discharges. I have frequently noted a peculiar abdominal eruption, in severe cases, which occurs as isolated dark, red spots, larger than those of typhoid fever, disappearing on pressure and with the decline of the fever, each lesion lasting about 24 hours.

In the *chronic form*, in addition to the diarrhea, emaciation and anemia are present. The stools are thin, watery, and numerous. The presence of undigested food in the evacuations indicates inflammation of the small intestine, while the presence of considerable mucus points to involvement of the large intestines.

Diagnosis.—*Colic* resembles enteritis only in the character of the pain and lacks abdominal tenderness, diarrhea and fever.

Typhoid Fever is distinguished by its prodromes, temperature record, eruption, enlarged spleen, character of the stools, and the Widal reaction.

Dysentery is characterized by small, mucous blood-stained discharges and marked tenesmus.

Peritonitis may be differentiated by its intense pain and tenderness, tympany, marked constitutional reaction, decubitus, and constipation.

Cholera may resemble enteritis when the attack is mild, but a bacteriologic examination of the stools will aid in making the diagnosis.

Prognosis.—The prognosis is favorable when the treatment is prompt and appropriate. Mild cases last for four or five days, severe cases may continue for one or two weeks. In chronic enteritis, the diarrhea may persist indefinitely.

Treatment.—The patient should be placed in bed and the diet restricted to such articles as milk, and lime-water, or mutton or chicken broths to which well-boiled rice has been added. In most cases it is well to begin the treatment by the administration of some mild laxative such as calomel, magnesia, or Epsom salt to relieve the intestinal tract of irritants. For adults, the best remedy is opium. The following formulas may be employed:

℞. Ext. opii ....gr. ¼-½ .016-.032 Gm.
Camphoræ pulv. ....gr. iij .2 Gm.
M. S.—In pill, every three hours.

Or—

℞. Tinct. opii deodorat.	♏ x	.6 Cc.
Liq. potassii citrat.	fʒ ij	8. Cc.

M. S.—Every hour until opium effect is manifested.

The strength and the frequency of administration of either of these formulas must be governed by the severity of the attack.

Salol, gr. i-iii (0.065-0.2 Gm.), alone or combined with bismuth salicylate, gr. x-xv (0.6-1 Gm.), every three hours may also be used.

If vomiting is annoying, all other treatment must be discontinued until it has been controlled, the following being usually efficient:

℞. Hydrargyri chlor. mit.	gr. ⅛	.008 Gm.
Sodii bicarbon.	gr. ij	.13 Gm.
Sacch. lac.	gr. ij	.13 Gm.

M. S.—Every hour or two, dry, on tongue.

For children:

℞. Tinct. opii deodorat.	♏ j	.06 Cc.
Bismuth. subnit.	gr. v	.32 Cc.
Mist. cretæ	fʒ j	4. Cc.

M. S.—Every two hours, for a child of one year.

If the disease shows the least tendency to linger, the acid treatment should be substituted, one of the best formulas being "Hope's Camphor Mixture." The following, which I have used with much success in the insane wards of the Philadelphia Hospital, where, at times, we see considerable intestinal catarrh, and which I have named "Mistura enterica," is generally satisfactory:

℞. Spts. camphoræ	f℥ j	30. Cc.
Acid. sulphurici dil.	f℥ iss	45. Cc.
Tinct. opii deodorat.	f℥ j	30. Cc.
Tinct. capsici	f℥ ss	15. Cc.
Spts. chloroformi	f℥ ss	15. Cc.
Spts. vini gallici q. s. ad	f℥ vj	ad 180. Cc.

M. S.—One to two teaspoonfuls, well diluted, every three or four hours.

In chronic cases, every attention must be given to the diet, hygiene, clothing, etc. Irrigation of the colon with silver nitrate solution (20 gr. to the pint) may be necessary. Mineral astringents and intestinal antiseptics are of great value.

Locally. Poultices, warm fomentations, such as turpentine stupe, or belladonna ointment or camphorated oil, are agreeable.

CROUPOUS ENTERITIS.

Synonym.—Membranous enteritis.

Definition.—A croupous inflammation of the mucous membrane of the small intestines; characterized by tenderness, paroxysmal pain, moderate fever, and the formation and discharge at stool of membranous shreds or casts.

Causes.—Adult life, female sex, neurotic temperament, hysteria, and hypochondriasis are the principal etiologic factors. A true croupous enteritis may occur in poisoning by inorganic substances, in the acute infectious diseases, and in the several cachexias.

Pathologic Anatomy.—A subacute inflammation of the small intestine, during which the mucous membrane becomes covered with a whitish or grayish-white, firmly adherent, membranous deposit, cemented together by a coagulable exudation, and prolonged by rootlets from the under surface into the intestinal follicles.

Symptoms.—The affection is manifested by paroxysms, each of which is preceded by various neurotic symptoms. The attack begins with feverishness, sense of soreness and distention of the abdomen, colicky pains of a spasmodic character, centering around the umbilicus and abdominal tenderness, which phenomena continue for one or two days. Diarrhea, pain, and tenesmus with the presence of mucus, shreds of membrane or cylindrical casts of the bowel, and sometimes blood in the stools then become manifest. Relief follows the discharge of the casts, although the generalized abdominal soreness may persist for a few days.

Diagnosis.—*Peritonitis* and *Dysentery* may resemble it in the early stage, but the mucous casts and membranous shreds which are passed within the first 48 hours, as a rule, will serve to make the diagnosis.

Prognosis.—Life is never threatened but the disease is very obstinate to treatment. The paroxysms occur at intervals of a week or two, but may be postponed for several months.

Treatment.—The underlying neurotic condition should receive the most careful attention. In addition the diet should be restricted so as to reduce the liquids to a minimum. The pain, which is sometimes excruciating, may require some preparation of opium, preferably morphin, hypodermically. The administration of an emulsion of castor oil and turpentine will aid in the expulsion of the cast and overcome any tendency towards constipation. Da Costa recommends some preparation of liquid tar for its alterative effect on the mucous membrane. Alteratives such as cod-liver oil, Fowler's solution of the arsenite of potassium, ℳ i-ii (0.06-0.12 Cc.), or bichlorid of mercury, gr. 1/6 (0.001 Gm.), may serve to prevent a return of the paroxysms.

CHOLERA MORBUS.

Synonyms.—Sporadic cholera; English cholera; cholera nostras; bilious cholera.

Definition.—An acute catarrhal inflammation of the mucous membrane of the stomach and intestines, of sudden onset; characterized by violent abdominal pains, incessant vomiting and purging, cold surface, rapid, feeble pulse, spasmodic contractions of the muscles of the abdomen and extremities, and prostration.

Causes.—Summer and early autumn season, sudden changes in the temperature, and the presence of irritants in the digestive tract such as result from the decomposition of food, and unripe fruit and vegetables seem to be the most important factors in its production. A special microörganism resembling the comma bacillus is often present in the stools.

Pathologic Anatomy.—Except in cases in which death has occurred within a few hours, the gastrointestinal mucous membrane is congested and denuded of epithelium and the solitary glands and Peyer's patches are swollen and prominent. The blood is thick, and dark in color; the kidneys are enlarged and congested; and in prolonged cases granular changes appear in the muscles.

Symptoms.—The onset is sudden and violent and often occurs after midnight being manifested by chilliness, intense nausea, vomiting, and purging accompanied by distressing burning or tearing abdominal pains or colic. The vomited matter at first consists of the ordinary contents of the stomach, but is soon replaced by bilious material and later, almost pure water. The stools are frequent and often continuous. At first, ordinary feces are discharged, then liquid whitish or greenish stools and later "rice-water" stools resembling those of Asiatic cholera. The surface of the body is cold and covered with clammy sweat, and in severe cases, intense muscular cramps are present. The pulse is small and feeble and there is intense thirst. Collapse may occur. The patient becomes rapidly weak and emaciated, the body appearing to shrink as in Asiatic cholera.

Diagnosis.—*Asiatic cholera* may resemble cholera morbus, but the history, and the presence of the comma bacillus of Koch in the stools of the former will serve to make the diagnosis.

Irritant poisons, such as tartar emetic and elaterium, produce symptoms that may be mistaken for cholera morbus and can only be distinguished by the history and the detection of the cause.

Prognosis.—The outlook is favorable in most cases. The mortality in all grades being about five per cent. Either extreme of life has an unfavorable influence. Mild cases may last only one or two days, but in the more severe cases the affection may persist for one or more weeks and be followed by a tedious convalescence.

Treatment.—In all cases, regardless of the cause, a hypodermic injection of morphin sulphate, gr. ⅛-⅓ (0.008-0.022 Gm.) and atropin sulphate, 1-120 (0.00054 Gm.), should be administered, to be repeated in a half hour if necesary. Liquid preparations of opium by the mouth or rectum may be occasionally necessary instead. Hope's camphor mixture may be used in some cases. The depression may be relieved by small doses of brandy or dry champagne. Small pellets of ice will overcome the intense thirst to some extent, but liquids are contraindicated.

If the vomiting and purging continue, the following may be made use of—

℞.	Bismuth. subnit. ....gr. xx	1.3	Gm.
	Acid. carbol. ....gr. ⅙	.01	Gm.
	Glycerini ....♏xx	1.3	Cc.
	Aquæ ....fʒ iv	15.	Cc.

M. S.—Every hour, in water.

If the vomiting is so severe that no opportunity occurs for the medicament to come in contact with the gastric mucous membrane, an enema of chloral, gr. x-xv (0.6-1 Gm.), in some demulcent with deodorized tincture of opium, ♏ x-xx (0.6-1.2 Cc.), acts often like magic in quieting the distress of the tortured patient.

For the muscular cramps Da Costa employs:

℞.	Chloral ....ʒ iv	15.	Gm.
	Petrolat. ....℥ j	32.	Gm.

M. S.—To be rubbed over the affected muscles.

Bartholow suggests—

℞.	Chloral	ʒ iij	12. Gm.
	Morphinæ sulph.	gr. iv	.26 Gm.
	Aquæ	f℥ j	30. Cc.

M. S.—Twenty minims, hypodermically, repeated p. r. n.

Locally, mustard poultices, turpentine stupes, and hot water-bottles to the abdomen will be of benefit.

The after-treatment will consist largely in regulation of the diet and a course of tonics.

ENTEROCOLITIS.

Synonyms.—Inflammatory diarrhea; ulcerative enterocolitis.

Definition.—A catarrhal inflammation of the lower portion of the small—ileum—and the upper portion of the large intestines, with a great tendency to ulceration of the intestinal glands if the catarrh becomes chronic.

Causes.—The affection is most common in childhood, particularly in the second summer. Improper and indigestible food, artificial feeding, summer season, impure air, uncleanliness, and exposure, are important etiologic factors. It may follow any of the infectious fevers and disorders of the intestinal tract such as diarrhea and cholera infantum. The *Bacillus dysenteriæ* of Shiga is often present in the evacuations.

Pathologic Anatomy.—The disease may be acute or chronic. In the *acute variety,* hyperemia, swelling edema, and softening of the mucous membrane of the ileum and upper part of the colon are present. The intestinal follicles are considerably hyperplastic, their excretory ducts being enlarged and tumid and readily distinguished as grayish or blackish points in the center of the glands. Peyer's patches present the same changes and a similar appearance, often seemingly ulcerated, but true ulceration is absent. In severe cases there may be pseudo-membranous formation.

In the *chronic variety,* the thickening and infiltration involves the submucous and muscular coats producing induration and rigidity of the intestinal walls. Ulceration occurs and extends through the entire thickness of the membrane. "These ulcers, when isolated, are from one to one and a half lines in diameter, oval or circular in shape, and either have sharp-cut edges, as though the piece of mucous membrane had been cut out with a punch, or the mucous membrane bounding them is undermined." The small ulcers often coalesce, so that large, irregular ulcerated patches of a grayish-white color are formed, having for their base the submucous or muscular coats. The mesenteric glands are enlarged, but seldom, if ever, undergo ulceration.

Symptoms.—The *acute form* may develop slowly with restlessness or fretfulness, or suddenly with feverishness, loss of appetite, thirst, nausea, vomiting, abdominal pain, and diarrhea. The abdomen soon becomes enlarged and tender. The stools are characteristic, being small, semifluid, heterogeneous, greenish, acid, and mixed with yellowish particles of ordinary feces and undigested casein which give to the evacuation the appearance of chopped spinach. They vary in number from fifteen to thirty in twenty-four hours. The temperature is irregular (102° to 104° F.) and the pulse-rate is increased. Emaciation is rapid and pronounced.

The *chronic form* usually follows the acute variety, the symptoms being less severe, but persistent. Loss of strength and emaciation become extremely pronounced. The temper is very irritable; the complexion grows dark, sallow, and unhealthy, and the face presents the "old man" appearance; the skin is dry and harsh, and, in consequence of the marked emaciation, either hangs in folds around the shrunken limbs or is drawn tightly over the joints; the abdomen is enlarged and tender, the stools numbering from six to a dozen during the day and night, consisting of the products of an imperfect digestion mixed with mucus, serum, pus, and oftentimes blood, having a semifluid consistency, and an extremely offensive odor. Ulcerative stomatitis is a frequent complication, adding to the discomfort of the patient. An irregular temperature record may occur with increased frequency of the pulse.

In fatal cases, the termination is ushered in with delirium, convulsions, stupor, coma, and other symptoms resembling hydrocephalus.

Diagnosis.—The distinctive features of this affection are the fever, abdominal distention and tenderness, emaciation, and the characteristic "chopped spinach" stools. *Cholera infantum* may be confused with it, but the rapid onset, high temperature, persistent vomiting, profuse serous stools, and early collapse in the former affection will serve to differentiate these conditions.

Prognosis.—Enterocolitis is always a serious affection. The acute cases usually subside in from ten days to two weeks, while the chronic forms last from one to three months or longer. Relapses are frequent. In vigorous children who have passed their first dentition the outlook is favorable, but in weak infants surrounded by unhygienic environments, the prognosis is grave. The prompt institution of appropriate treatment favorably influences the prognosis.

Treatment.—The feeding should be first temporarily withheld and afterwards altered to suit the individual needs of the patient. When possible, a change of air with cleanliness and rest is desirable. The intense suffering of the little patients calls for anodynes and the progressive emaciation indicates the use of whiskey or brandy (10 to 20 minims) every three or four hours. The following formulas may also be used with advantage:

℞.	Salol	gr. ij	.13 Gm.
	Bismuthi subnit.	gr. v	.32 Gm.

M. Ft. chart. No. i.

S.—Such a powder every two hours.

Or—

℞.	Hydrargyri chlor. mit.	gr. ss	.032 Gm.
	Pulv. ipecac.	gr. ss	.032 Gm.
	Pulv. opii	gr. ss	.032 Gm.
	Cretæ præparat.	gr. xx	1.3 Gm.

M. Ft. chart. No. xij.

S.—One every two or three hours, to child of one year.

The compound kino powder, lactic acid, and subnitrate of bismuth in small, but frequently repeated doses may be of benefit.

The following is a good combination:

℞.	Bismuthi subnit.	ʒ iij	12. Gm.
	Tinct. kino	fʒ iss	45. Cc.
	Tinct. opii camphorat.	fʒ iss	45. Cc.
	Mist. cretæ	℥ iij	90. Cc.

M. S.—Tablespoonful every few hours.

Flushing of the colon with cold normal salt solution, or solutions containing silver nitrate (one grain to the ounce), tannic acid (five grains to the ounce), or sodium benzoate (four grains to the ounce) is an extremely valuable part of the treatment. For tenesmus, Rotch advises suppositories containing one quarter grain of cocain. The abdominal pain and distress may be relieved by the application of hot water-bottles, mustard plasters, turpentine stupes, or the spice poultice. The spice poultice is made up of a half-ounce (15.6 Gm.) each of cloves, allspice, cinnamon, and anise seeds, which are pounded together in a mortar and placed between two pieces of coarse flannel about six inches square. This should be soaked in equal parts of hot whiskey or brandy and water and then applied to the abdomen, being again heated as it becomes cool.

The *chronic form* differs but slightly in its treament from the acute form. The diet requires considerable attention to alter and predigest the artificial foods to meet the various indications. Fresh air, salt baths, cleanliness, and other details of hygiene demand consideration. Among drugs, bismuth, pepsin, and salicin will be found of benefit. The following formulas may also be employed:

℞. Argenti nitrat. ....gr. j	.065 Gm.
Acid. nitric. dil. ....♏xv	1. Cc.
Mucil. acaciæ ....f℥ ss	15. Cc.
Aq. cinnamomi .....ad f℥ iij	ad 90. Cc.

M. S.—Teaspoonful, diluted, every three of four hours.

Or—

℞. Acidi carbolici ....gr. 1-12-⅛	.005-.008 Gm.
Tincturæ iodi ....♏j-ij	.06-.12 Cc.
Aquæ menthæ pip......fʒ j	4. Cc.

M. S.—Every three or four hours.

Or—

℞. Tinct. calumbæ ....fʒ iij	12. Cc.
Liq. ferri nitratis.......♏xxx	2. Cc.
Syrupi zingib....q. s. ad f℥ iij	ad 90. Cc.

M. S.—One or two teaspoonfuls, according to age, every three or four hours, diluted.

Or—

℞. Quininæ muriat. ....gr. xxv	1.6 Gm.
Acid. tannici ....gr. x	.6 Gm.
Syr. limonis...........fʒ ij	8. Cc.
Aq. chloroformi.....ad fʒ iij	ad 12. Cc.

M. S.—Teaspoonful every two hours.

CHOLERA INFANTUM.

Synonyms.—Choleriform diarrhea; summer complaint.

Definition.—An acute catarrhal inflammation of the mucous membrane of the stomach and intestines, together with an irritation of the sympathetic nervous system, occurring in children during their first dentition; characterized by severe colicky pains, vomiting, purging, febrile reaction, and prostration.

Causes.—Hot weather, infancy, dentition, improper food, bad hygiene, and constitutional predisposition are the most important causes. The exciting cause is probably some specific microörganism or its toxin. This affection constitutes from two to three percent of the summer complaints of children.

Pathologic Anatomy.—There are no characteristic lesions, the gastrointestinal mucous membrane is, however, usually the seat of catarrhal inflammation.

Symptoms.—The onset is sudden, being ushered in with vomiting, purging, abdominal pain, high fever (105°-106° F.) rapid pulse, and intense thirst. The vomited matter consists of partly digested food, sero-mucus, and finally bilious material. Distressing retching accompanies the vomiting. The tongue is coated. Thirst is a marked feature of the disease, and ice and water will be taken incessantly for its relief only to be rejected a few minutes later. The stools are first partly fecal; but soon become watery or serous, soaking the clothing and leaving a faint greenish or yellowish stain. They number from ten to twenty a day and possess a musty and at times fetid odor. The temperature should be taken in the rectum, as the surface temperature is comparatively low. The pulse is rapid and feeble, ranging from 130 to 160 per minute. These various symptoms continue but a few hours before rapid wasting ensues. The body shrinks, the eyes are shrunken and partly closed, the mouth partly open, and the lips are dry, cracked, and bleeding. The child is at first irritable and restless, but soon passes into a semicomatose condition, the pulse becoming more and more feeble; the body-surface is cold and clammy; the pupils contract, but are irresponsive to light; and the stupor deepens. The termination may be in death with profound exhaustion or convulsive seizures, or in recovery, the symptoms gradually ameliorating and passing into a slow and tedious convalescence.

Diagnosis.—The characteristics of cholera infantum which serve to distinguish it from other enteric affections are the rapid onset, the constant serous vomiting and purging, the intense thirst, the high fever, prostration, and rapid emaciation.

Prognosis.—The outlook is unfavorable. Many cases end by collapse within twenty-four to forty-eight hours. The choleraic symptoms never last more than five days, and if the patient survives this period, recovery after a protracted convalescence is probable. Relapses are common.

Treatment.—The first indication is to thoroughly empty the digestive tract by washing out the stomach and irrigating the bowel with cold water. Morphin, gr. 1-100 (0.00066 Gm.), and atropin, gr. 1-500 (0.000132 Gm.), may be given hypodermically to a child one year old, to combat the nervous and cardiac symptoms. Normal salt solution should be administered hypodermically or by the bowel. The fever will require cool bathing, or sponging with alcohol and water, and the application of an ice-bag to the head. In the early stages it is best to withhold feeding; later brandy, ♏ v-x (0.3-0.6 Cc.) and barley water should be given every hour. If the stomach is absolutely unretentive, the stimulation should be administered hypodermically. The vomiting may be controlled to a greater or less extent by large doses of bismuth or chloral, gr. i-iii (0.065-0.21 Gm.) by the mouth in a demulcent, or double the quantity by the rectum, or one of the following:

℞.	Bismuth. subnit.	ʒ ij	8.	Gm.
	Acid. carbolici	gr. j	.065	Gm.
	Mist. acaciæ,			
	Aq. menth. pip......	āā f℥ j	āā 30.	Cc.

M. S.—Teaspoonful every half-hour, hour, or two hours.

Or—

℞.	Hydrargyri chlor. mit....	gr. 1-20	.003	Gm.
	Bismuth. subnit.	gr. ij-v	.13-.3	Gm.

M. S.—A powder every half hour.

Benefit may also be obtained from the use of bismuth salicylate, gr. ii (0.13 Gm.) with sugar of milk every hour or two, or salol, gr. i-ii (0.065-0.13 Gm.) every two or four hours. When depression supervenes the feeding should be every two hours; water or ice should be given to quench the thirst; and cognac brandy, ♏ v-x (0.3-0.6 Cc.), should be administered every hour or two by the mouth, rectum, or hypodermic injection. In the event of collapse, the hot bath should be employed and a hypodermic injection of strychnin (gr. 1-100 or 0.00066 Gm. to a one-year old child) should be given. The nervous symptoms when marked may require potassium bromid or valerian.

Locally, the application of hot water-bottles, mustard or spice poultice, or turpentine stupes to the epigastrium will afford relief.

During convalescence a change of air is of great benefit. Every detail of the hygiene should be improved. Peptonized milk should be given for a long period, substituted occasionally by barley-water, albumin-water, and fresh beef-juice. The feeding should be carefully watched and modified from time to time as the occasion arises.

ACUTE DYSENTERY.

Synonyms.—Colitis; ulcerative colitis; bloody flux.

Definition.—An acute inflammation of the mucous membrane of the large intestines; either catarrhal or croupous in character, followed in some cases with ulceration, characterized by fever, tormina, tenesmus, and frequent, small, mucous, and bloody stools. It may be sporadic, endemic, or epidemic and occurs in four clinical forms: acute catarrhal, amebic or tropical, croupous or diphtheritic; and chronic dysentery.

Causes.—The predisposing causes of all forms are summer and autumn season, warm climate, sudden atmospheric changes, errors in diet, impure drinking water, exposure to cold and wet, cachectic states, and bad hygiene.

The catarrhal form is usually brought about by the ingestion of irritating food. It is sporadic and is not associated with any specific microörganism. It frequently accompanies the infectious fevers. This form, if prolonged, constitutes chronic dysentery.

The amebic or tropical dysentery is due to the presence of the *Amœba coli* in the colon. The organism is from 15 to 20 microns in diameter and consists of a central portion of granular protoplasm surrounded by a narrow zone of clear protoplasm. The organism may be found in the stools, in the ulcerations of the colon, and in the hepatic abscesses that not uncommonly result. This variety of the disease may be sporadic or epidemic.

Croupous, diphtheritic, or bacillary dysentery is due to the *Bacillus of Shiga,* a microörganism belonging to the colon-typhoid group of bacilli possessing flagella and motility. It produces an agglutination reaction with the blood of dysenteric patients similar to the Widal test. This variety of the disease is common in temperate and tropical regions, occurring as pseudomembranous, croupous, ulcerative, and chronic dysentery. It may be epidemic or sporadic.

Dysentery is not contagious in the ordinary sense of the word, but is infectious, the drinking-water being the usual medium of infection.

Pathologic Anatomy.—In the *catarrhal form,* the mucous membrane and submucous coat of the colon are swollen, congested, and edematous, and mucus is formed in excess. The follicles are enlarged and may become ulcerated.

In *amebic or tropical dysentery,* the lesions are situated also in the colon, but may be found in the ileum. Ulceration, involving the mucosa and submucosa, is the characteristic structural change. This process is preceded by the infiltration of the mucous and submucous coats with a grayish, gelatinous substance, the exfoliation of which produces the ulcer. In the early stages these local infiltrations appear as hemispheric elevations, the mucous membrane covering, which is soon cast off to be followed by sloughing of the submucous coat and its infiltrate. The microörganisms are present in the necrotic tissue and by their migration not infrequently (20 percent) produce abscess of the liver.

Croupous or *diphtheritic dysentery* begins with intense congestion, swelling, and edema of the mucous and submucous tissue, with extravasations of blood and the whole mucous membrane is covered with a firm, fibrinous exudation; the mucous membrane softens and sloughs, leaving large ulcers and gangrenous spots. If recovery occurs, large cicatrices form, which narrow the caliber of the intestinal tube. The mesenteric glands enlarge, and soften, and abscesses form in them; the liver becomes the seat of small abscesses, from embolic obstruction of the radicles of the portal vein; the heart muscles are flabby and more or less fatty.

Symptoms.—The *catarrhal form* begins gradually, with diarrhea, loss of appetite, nausea, and very slight fever, which continues for two or three days, when the true dysenteric symptoms develop, to wit, pain on pressure along the transverse and descending colon, tormina or colicky pains about the umbilicus, burning pain in the rectum, with the sensation of the presence of a foreign body and a constant desire to expel it, or tenesmus. The stools for the first day or two contain more or less fecal matter, but they soon change to a grayish, tough, transparent mucus, containing more or less blood and pus. The number of stools varies from five to twenty or more in the twenty-four hours. During the tormina, nausea and vomiting may occur. The urine is scanty and high-colored. The duration is about one week, the patient rapidly becoming emaciated and enfeebled.

The *amebic form* is characterized by a more gradual onset and gradually increasing diarrhea. The stools are frequent, bloody, mucoid, and very fluid, but as the disease progresses they become yellowish gray and contain mucous and sometimes blood. The stools are less in number and the tenesmus is not so great as in the preceding variety. Actively moving *Amœbæ coli* are found in the evacuations, disappearing as the stools become formed. Fever is not very high and may be absent. Emaciation is marked. Abscess of the liver and lungs may occur as complications. The duration varies from six to twelve weeks and convalescence is protracted. Periodic recrudesences are not uncommon.

The *croupous variety* has an acute onset. The stools are more frequent and contain more blood and pus, patches of membrane, sometimes casts of the bowel, and portions of the gangrenous mucous membrane. Nausea, vomiting, and great prostration and emaciation are present. The skin is cold; the pulse is feeble; and the odor emanating from the patient is fetid. Gaseous distention of the abdomen is common. The fever is high; the tenesmus is severe; and the adynamia is profound. The *Bacillus of Shiga* is present in the discharges. The duration of the grave symptoms is three or four days, when collapse and death occur or protracted convalescence begins. Hepatic abscess, intestinal perforation, arthritis, and paralysis may occur as complications. This variety of the disease may become chronic and may occur in the course of heart, lung, or kidney disease.

Chronic Dysentery.—A persistence in the intestinal lesions of any of the acute varieties just described results in chronic dysentery. Ulceration is present in most cases, but in others the intestinal walls are thickened with scattered slate-colored patches of blood extravasation and disintegration. Diarrhea and emaciation are the principal symptoms. Abdominal pain and tenesmus are slight. Acute exacerbations are frequent. The affection may last several months or even years.

Diagnosis.—The blood-stained stools, tenesmus, abdominal pain, and the history will aid in distinguishing dysentery from other enteric conditions. The variety of the disease may be recognized by the microörganisms in the stools, and the symptoms.

Acute catarrhal enteritis is not attended by tenesmus, or blood-stained mucoid stools.

Malignant rectal disease is attended by blood-stained stools and tenesmus and resembles chronic dysentery, but an examination will serve to clear up the diagnosis.

Intussusception is accompanied by mucoid and bloody stools with tenesmus, but the abrupt onset, persistent vomiting, and the presence of a sausage-shaped tumor in the abdomen will distinguish it from dysentery.

Treatment.—The patient should be confined to bed in even the mildest attack, and the bed-pan employed, being careful to thoroughly disinfect the discharges with ferrous sulphate or chlorinated lime. The diet should be bland and unirritating. Substances such as milk and lime-water, beef-peptonoids, broths, egg-albumin, etc., may be given in acute attacks. A semisolid diet is permissible in chronic cases. The medicinal treatment should be begun by the administration of a purgative, preferably castor oil, f℥ (30 Cc.), with tincture of opium, gtt. x-xx (0.66-1.33 Gm.). When there is high fever and no marked evidence of adynamia, magnesium sulphate ʒii (8. Gm.) or Rochelle salt ʒiv (16. Gm.), may be given in water every hour until there is copious purgation. (Saline Treatment).

The pain, tenesmus, and peristalsis will require opium in some form, alone or combined with astringents. A hypodermic injection of morphin sulphate, gr. ¼-½ (0.016-0.032 Gm.), given every three or four hours as required is very efficient.

℞.	Ext. opii ....gr. ss	.032 Gm.
	Plumbi acetat. ....gr. ij	.13 Gm.

M. S.—Every two hours.

Or—

℞.	Pulv. opii	gr. ss	.032 Gm.
	Plumbi acetat.	gr. ij	.13 Gm.
	Pulv. ipecac.	gr. ¼	.016 Gm.

M. S.—Every two hours, until character of stools change.

I have frequently seen the character of the stools change within twenty-four hours with the following, which I call *Mistura enterica*, viz.:

℞.	Acid sulph. dil.	f℥ ss	15. Cc.
	Tinct. opii deodorat.	f℥ j	30. Cc.
	Spts. camphoræ	f℥ j	30. Cc.
	Tinct. capsici	f℥ ss	15. Cc.
	Spts. chloroformi	f℥ ss	15. Cc.
	Spts. vini gallici	f℥ iss	45. Cc.

M. S.—One teaspoonful every two or three hours, diluted.

In strong young individuals the very best prescription possible is—

℞.	Magnesii sulph.	ʒ j	4. Gm.
	Acid. sulph. dil.	♏x	.6 Cc.
	Tinct. opii deodorat.	♏x	.6 Cc.
	Aquæ chloroformi	ad ʒ ij	ad 8. Cc.

M. S.—To be given every two or three hours until feces appear in the stools when small doses of opium and quinin sulphate may be used.

Hope's original camphor mixture at times acts favorably:

℞.	Acidi Nitrosi	fʒ i	4. Gm.
	Mist. camphoræ	f℥ viii	240. Gm.
M. et adde			
	Tr. opii	gr. xl	1.2 Gm.

M. S.—One-fourth of this mixture to be taken every three or four hours.

Bismuth subnitrate, gr. xxx (2 Gm.), or bismuth salicylate, gr. xx (1.3 Gm.), every two or three hours is of value. Loomis recommends ipecacuanha, gr. ¼ (0.016 Gm.) every half-hour with sufficient opium to secure quietness. The East Indian physicians employ it in amebic and croupous dysentery in large doses, (20 to 60 gr.-1.332-4 Gm.). Its administration is preceded by a dose of tincture of opium one-half hour before and for three hours after, only milk and iced soda-water in small quantities are allowed. On the second day the dose of ipecacuanha is reduced and the drug is combined with intestinal antiseptics.

In children the following combination is successful:

℞.	Pulv. ipecacuanhæ .....gr. ¼	.016	Gm.
	Bismuth. subnit. ....gr. v-x	.32-.65	Gm.
	Cretæ præp. ....gr. iij	.2	Gm.

M. S.—Every two hours.

Ringer advocates the use of bichlorid of mercury, gr. 1-100 (0.00065 Gm.), every hour or two, claiming that it soon frees the evacuation of blood and slime. Nuclein (Aulde), gr. i (0.065 Gm.) every hour until the character of the stools changes is also of value.

Serum Treatment.—The antidysenteric serum obtained from the horse after immunization, has been employed on animals in the laboratory with success. Shiga has used it in 250 cases of Japanese dysentery with a mortality of about 10 percent, which, under the ordinary methods of treatment, would have been about 36 percent.

Irrigation of the rectum with either tepid, hot, cold, or iced water adds greatly to the patient's comfort and to the decrease of the inflammatory process. A one to two percent solution of creolin may be used. Osler employs warm injections of quinin, 1 to 5000, 1 to 2500, in amebic dysentery with great benefit and rapid destruction of the amebæ. Suppositories of ice, iodoform, or opium will afford great relief, lessening the pain and tenesmus.

Poultices, stupes, hot water-bottles, etc., may be applied over the abdomen, but are seldom very beneficial.

Chronic dysentery will require careful modification of the diet and rest in bed. Internally, bismuth subnitrate, gr. xxx (2. Gm.), three times daily; turpentine, ♏x (0.6 Cc.) every three hours; silver nitrate, gr. ⅛-⅓ (0.008-0.022 Gm.), three times daily; sulphur, gr. x (0.6 Gm.), three times daily; or the following may be administered:

℞.	Cupri sulphat. ....gr. ⅙	0.011	Gm.
	Ext. opii (aq.).........gr. ¼	0.016	Gm.
	Ext. nucis vomicæ......gr. ⅙	0.011	Gm.

M.—Ft. pil. No. i.

S.—To be taken four times daily.

Cases which are continued by reason of ulcerated patches in the colon require intestinal irrigations. Silver nitrate, gr. x to xxx (0.6 to 2. Gm.) to the pint is the solution of selection. The patient should be placed on the back with the hips elevated. The preliminary injection of a small quantity of cocain (4 percent solution) will relieve any irritability of the rectum. The irrigating solution is allowed to flow into the bowel through a long rubber tube connected to a fountain syringe, the bag portion of which is elevated. The injection should be made from two to three times a week, employing from one to three pints or more of the solution. Alum, sulphate of zinc, acetate of lead, or copper sulphate may be used instead of silver nitrate in solution. The following is sometimes given by injection:

℞.	Argent. nitrat. ....gr. i	0.065 Gm.
	Tr. opii deodorat........♏xv	1. Gm.
	Aquæ amyli ....f℥ iv	120. Gm.

M. S.—Use as directed.

During convalescence, the internal administration of cod-liver oil, syrup of the lactophosphate of lime, and the following combination will be required:

℞.	Strychnin. sulph. ....gr. ss	0.032 Gm.
	Acid. hydrochlor. dil....fʒ ii	8. Gm.
	Tr. gentian. comp. q.s.ad f℥ iv	120. Gm.

M. S.—One teaspoonful in water before meals.

TYPHLITIS.

Synonyms.—Inflammation of the cecum; typhlitis stercoralis.

Definition.—A catarrhal inflammation of the mucous membrane of the cecum and ascending colon; characterized by pain, tenderness, constipation, and in certain cases a characteristic vomiting.

Causes.—I do not believe the term "typhlitis" is to be supplanted by the term appendicitis; I am convinced there are two conditions having some symptoms in common. In a majority of instances typhlitis is mechanical, due to the accumulation of feces in the cecum.

Symptoms.—Pain and tenderness in the right iliac region and over the ascending colon with prominence of this portion of the abdomen are among the early manifestations. The temperature rises and in a certain number of cases an eruption of one or two pin-head sized, dark-red spots appears on the abdomen. The bowels are distended with gas and constipation is usually present, but small liquid stools may occur at times due to the accumulation of hardened feces in the sacculated periphery of the cecum, leaving a central canal through which the liquid contents of the upper bowel may pass.

In very severe cases all the symptoms are intensified. Constipation becomes greater; no flatus is discharged; and the feces become impacted. The fever is high, and restlessness, nausea, and vomiting supervene. The vomit consists first of the stomach-contents, then those of the duodenum, with bilious matter, and ultimately fecal material, if impaction has taken place. Profound depression accompanies these symptoms. Peritonitis may develop from rupture of the intestine or by contiguity of structure.

Diagnosis.—The constipation, localized pain, tenderness, and prominence, and tympanites will serve to distinguish it from appendicitis and other intestinal affections. The history, and the foregoing distinctive features will separate it from intestinal obstruction.

Prognosis.—The mild form is favorable, usually lasting but one week. The severe form is grave, but not necessarily fatal. It may end in subacute peritonitis and be prolonged two weeks or more.

Treatment.—The patient should be placed in bed and the diet restricted to milk in small amounts. The accumulation of feces may be prevented and the consequent inflammation retarded by the use of the following:

℞.	Magnesii sulph. ....ʒ xij	48. Gm.
	Acid. sulphurici dil.....fʒ ij	8. Cc.
	Tinct. opii deodorat.....fʒ iv	15. Cc.
	Spts. chloroformi ......fʒ ij	8. Cc.
	Aquæ menth. pip., q.s.ad f℥ iij	ad 90. Cc.

M. S.—One teaspoonful every hour, diluted.

A specific action is claimed for calomel by some observers, and may be thus employed:

℞.	Hydrargyri chlor. mit...gr. ij	.13 Gm.
	Sodii bicarb. ....gr. xx	1.3 Gm.
	Sacc. lac. ....gr. xxx	2. Gm.

M.—Ft. chart. No. x.

S.—One every hour until twelve have been taken, followed by hot Hunyadi Janos water or other saline purgatives.

When the pain is severe morphin and atropin should be administered hypodermically. If impaction is feared, the bowel should be irrigated with warm soapsuds and sulphate of magnesia should be given in one dram doses every two hours. Heat or cold may be applied over the abdomen.

PERITYPHLITIS—APPENDICITIS.

Synonyms.— Peritpyhlitic abscess; suppurative appendicitis; pericecal abscess; iliac abscess.

Definition.—*Perityphlitis;* an acute inflammation of the connective-tissue around the cecum (with localized peritonitis) leading to the formation of an abscess.

Appendicitis.—An acute or subacute inflammation of the appendix vermiformis, involving the surrounding tissues (with a localized peritonitis), leading to perforation of the appendix and the development of an abscess.

Causes.—Fecal impaction, foreign bodies, errors in diet, acute indigestion, exposure, intestinal catarrh, male sex, early adult life and the peculiar anatomy of the appendix are the principal predisposing causes. The exciting cause is a microörganism in all probability the *Bacillus coli communis* but *streptococci, staphylococci,* and the *Proteus vulgaris* have been associated with it. It may follow tuberculosis, typhoid fever, or influenza, in which cases the exciting cause is probably the bacterium which produced the preceding infectious fever. Torsion of the appendix may be a cause.

Pathologic Anatomy.—The inflammation of the appendix may be catarrhal, ulcerative, or intersititial.

Catarrhal appendicitis consists in a desquamative inflammation of the mucous membrane, which becomes swollen and sometimes obliterates the lumen of the tube. In some cases the excoriated surface becomes the avenue of infection and the disease terminates in an acute infectious peritonitis.

Ulcerative appendicitis is characterized by varying grades of ulceration of the mucous membrane and submucous tissue, and may terminate in perforation. It is not infrequently associated with fecal concretions and foreign bodies. Typhoid and tubercular ulcerations may be encountered.

Interstitial appendicitis may have its origin in an abraded or ulcerated surface of the mucous membrane or it may arise independently in the structure of the appendix wall, the infection being carried by the lymphatics. It is extremely virulent and is commonly associated with necrosis or gangrene of the appendix wall, thereby leading to perforation and a consequent virulent type of peritonitis. It may terminate fatally before the necrosis becomes manifest.

In all forms there is a localized or generalized peritonitis, which, by its resultant adhesions, aims to wall off the infection from the general peritoneal cavity. In the mild forms this is accomplished to a great extent, but in severe cases in which pus forms and the appendix ruptures, these adhesions for a time form the walls of an abscess, but they, too, ultimately rupture, discharging the contents of the abscess into the peritoneal cavity, bowel, bladder, vagina, or externally. Sometimes the exudate into the tissues surrounding the appendix is absorbed.

Symptoms.—The affection begins with a feeling of weight and soreness and rapidly developing severe pain over the entire abdomen, but most marked in the right iliac region. The pain is increased by coughing, deep breathing, and by lying on the left side, so that for relief the right leg is drawn up and the *dorsal decubitus* assumed. Localized tenderness accompanies the pain and corresponds to the situation of the diseased structure. Usually it may be detected by palpation at a point midway between the umbilicus and the anterior superior spine of the ilium (McBurney's point). In the early stage, there is rigidity of the right abdominal rectus muscle and adjacent muscles, which rigidity is replaced in two or three days by an oval tumor, usually about the size of a hen's egg, lying in the right iliac region, parallel to Poupart's ligament. Percussion over this enlargement yields impaired resonance or dullness. Occasionally the note is normal. Nausea and vomiting are frequent and often occur early in the attack. The tongue becomes coated and the appetite is lost. Constipation is the rule, but it may be replaced by diarrhea. Fever (102° to 104° F.) is present from the onset and may or may not be preceded by a chill. There is a corresponding increase in the pulse-rate. Suppuration is usually manifested by irregular fever and chills, sweats, and a feeling of tension or throbbing in the region of the appendix, but may be unattended by fever. Gangrene of the appendix may occur in the presence of a normal temperature. A sudden fall in the temperature usually indicates perforation of the structure. The urine has the characteristics of fever urine, and in addition contains a large quantity of indican. Leukocytosis is present in most cases.

Complications.—Local or general peritonitis, perforation, and abscess formation are the most common complications. Localized peritonitis gives rise to adhesions which may produce intestinal obstruction. Generalized peritonitis may result from extension of the inflammation or rupture of the appendix. Suppuration of the appendix may be followed by generalized peritonitis, hepatic abscess, lumbar abscess, perinephritic abscess, or multiple pyemic abscesses. Chronic appendicitis, in which the attacks recur at intervals, is a common sequel in cases in which the appendix is left undisturbed.

Diagnosis.—It differs from *typhlitis* in the absence of the colicky pains, dyspeptic symptoms, costiveness, and tympanites preceding the development of a tumor.

Typhoid fever is distinguished from appendicitis by its more gradual onset, the characteristic temperature record, diarrhea, enlargement of the spleen, rose-colored abdominal rash, and the Widal reaction.

Intestinal obstruction is unattended by fever, there is no localized tenderness, constipation is more complete, the pain is diffuse, and the vomiting may be stercoraceous.

Rectal growths, tubal disease, and *ovarian tumors* may be recognized and differentiated from appendiceal inflammation by physical examination by rectum and vagina.

Acute indigestion is characterized by an absence of localized pain and tenderness. Diarrhea is common and there is no enlargement in the right iliac region.

Hepatic colic is attended by jaundice and intermittent pain higher up in the abdomen extending to the right shoulder. Fever is usually absent.

Nephritic colic is marked by an absence of fever and localized rigidity, and by paroxysmal pain extending from the lumbar region into the groin and testicle.

Hepatic and Renal Abscesses may be distinguished by their location and character of the pain. They often occur as the result of a suppurating perityphlitis and frequently are only recognized after the abdomen has been opened.

Prognosis.—The outlook depends entirely on the character of the disease and the treatment. In non-suppurative cases recovery is the rule. Suppurative cases, in which surgical treatment has been instituted, show a mortality of about 25 percent. The mortality is 75 percent in the presence of generalized peritonitis. In operations between the attacks the mortality is less than one percent.

Treatment.—As soon as the diagnosis has been made the case should be placed in charge of a surgeon as it is difficult to predict the termination even in apparently mild cases. The patient should be placed at rest in bed and the diet restricted to liquids. In the early stages a mild laxative, such as castor oil, calomel, or citrate of magnesia may be administered. In advanced cases purgation may induce perforation. Enemas may be cautiously used. Heat or cold applied to the abdomen will, in a measure, relieve the pain. Morphin, hypodermically, may be necessary, but it should be remembered that it masks important symptoms. If the symptoms do not subside under this plan of treatment within 24 or 48 hours, it is customary to resort to a surgical operation. Some surgeons operate as soon as the diagnosis is made. In mild cases in which the symptoms abate and the attack is the first one, the operation may be postponed until the interval between the attacks. If the symptoms become less in severity, but do not entirely subside, operation is indicated at once. Complicated cases also require immediate operation. No definite rules can be followed in appendicitis, each case possessing features which place it in a class by itself. In cases in which it is certain an operation will not be performed, counterirritation may be applied to the abdomen.

PROCTITIS.

Synonyms.—Catarrh of the rectum; dysentery; rectitis.

Definition.—A catarrhal inflammation of the mucous membrane of the rectum and anus; characterized by pain, tenesmus, and frequent stools of hardened feces, or of mucus, pus, and blood.

Causes.—It may arise from constipation, habitual use of enemas and purgatives, diseases of the liver, hemorrhoids, and sitting on damp ground or stone steps.

Symptoms.—Burning pain in the rectum, tenesmus, the passage of hardened feces, or stools containing mucus, muco-pus, or blood, and prolapse of the mucous membrane are the most prominent symptoms. Nausea, headache, feverishness, and malaise may be present. In severe cases strangury and vesical tenesmus may be present, and periproctitis and fistulas may occur if the affection is protracted. Hepatic abscess and peritonitis may arise as complications.

Diagnosis.—Physical examination of the rectum will serve to distinguish it from hemorrhoids and uterine displacements, which are somewhat similar as regards their symptomatology.

Prognosis.—The outlook is favorable in uncomplicated cases.

Treatment.—As constipation is the most common cause it should be relieved by a soap and warm water enema, rectal irrigations, or the following injection:

℞. Magnesii sulph.	℥ ij	60. Gm.
Glycerini.	f℥ ss	15. Cc.
Aquæ bul., ...	f℥ iv	120. Cc.

M. S.—Use as directed.

Glycerin may be employed in suppository or enema, or the following emollient enema may be used:

℞. Ol. olivæ, ...	f℥ ij	60. Cc.
Tinct. opii deodorat., ...	♏xv	1. Cc.

M. S.—Use as directed.

Hot injections of strong black coffee, using from a half-pint to a quart, are valuable in irritability of the rectum with a tendency to diarrhea. Occasionally cold injections are more beneficial.

If periproctitis and suppuration supervene early incision is indicated.

INTESTINAL OBSTRUCTION.

Synonyms.—Intestinal occlusion; strangulated hernia; invagination; intestinal stricture; ileus.

Definition.—A sudden or gradual closure of the intestinal canal; characterized by pain, nausea, vomiting, constipation, and finally collapse.

Causes.—The numerous causes are arranged as follows:

1. *Accumulations within the bowel* of hardened feces, or foreign bodies.

2. *Strictures* the result of cancer, ulceration, or cicatrices.

3. *Pressure against the bowel,* from peritoneal adhesions, tumors, and abnormal growths.
4. *Strangulations,* due to the numerous forms of hernia.
5. *Invagination* or intussusception, the most common.
6. *Twisting,* volvulus or rotation of the bowel.

Pathologic Anatomy—*Invagination* calls for special description. It is usually caused by the lower portion of the ileum slipping down into the cecum, as the finger of a glove might be invaginated, causing thus an actual mechanical obstruction; this is produced by a spasm of the ileum, whereby its caliber is greatly diminished, thus permitting its descent into the lower bowel. Resulting from this occlusion or compression, are congestion, inflammation, with secondary constitutional reaction and death, or more rarely the invaginated bowel sloughs off and is voided by stool, union taking place at its site and recovery following.

Symptoms.—In *acute cases,* sudden spasmodic abdominal pain which soon becomes continuous in character is an early symptom. Constipation, which is unrelieved by purgatives or enemas, is present. The abdomen becomes greatly distended and very tender in spots. Nausea and vomiting occur; the vomit persisting and at length becoming stercoraceous. As the condition progresses, pinched features, sunken eyes, quick, feeble pulse, cold, clammy skin, and other symptoms of collapse become manifest. The duration of this form is about a week or ten days, when death may occur, or more rarely the symptoms may subside and there is a gradual return to health.

In *chronic cases* obstinate constipation, with the passage of ribbon-shaped stools or scybalous masses, abdominal pain and distention, and failure of health are the principal symptoms. The onset is gradual. It may become acute when the obstruction is complete. In rare instances small, fecal, muco-purulent stools, containing more or less blood, are passed.

Diagnosis.—The features of intestinal obstruction that are of most value in making a diagnosis are the obstinate constipation, the early vomiting, which shortly becomes stercoraceous in character, the abdominal distention, the absence of any discharge of flatus by the bowel, and early collapse. The X-ray may aid in locating the obstruction.

Acute peritonitis resembles intestinal obstruction to some extent, but the fever, diffuse tenderness, and the absence of a tumor and fecal vomiting point to peritonitis.

Strangulation in hernia is attended by the same symptoms as intestinal obstruction, and in the event of their occurrence the various abdominal rings should be carefully examined. Sometimes internal strangulation results from a portion of the intestine slipping through the foramen of Winslow, the diaphragm, or a slit in the omentum or mesentery, or under Meckel's diverticulum or inflammatory adhesions.

The situation of the obstruction may be indicated by the presence of a tumor. Fecal vomiting usually points to obstruction of the small intestine. Active peristalsis is always present a short distance above the obstruction.

The nature of the obstruction in many instances may be determined indirectly. In the large intestine more than one-half the cases of obstruction are due to intussusception, about one-third to twists, and about one-eighth to stricture and tumors. In the small intestine nearly three-fourths of the cases result from strangulation, about one-sixth from gall-stones, and about one-twelfth from intussusception. In children intussusception is the most common cause of obstruction. It may be recognized by the sausage-shaped tumor along the colon, and by digital examination by the rectum. Obstruction, due to twists, malignant growths, and strictures is usually low down and may be detected by rectal examination. The history of attacks of peritonitis will point to inflammatory adhesions as the cause of the obstruction. Alteration in the thoracic percussion note will indicate the passage of a portion of the bowel through the diaphragm. Marked meteorism in the right inguinal region is considered to be a diagnostic symptom of obstruction by Meckel's diverticulum. Fecal impaction is distinguished by its gradual onset and course, the history, and of an irregular tumor along the line of the colon.

Prognosis.—The prognosis is always grave but is most favorable in fecal impactions. In invagination the outlook is less favorable, but recoveries occur; the longer the symptoms continue the more favorable becomes the prognosis. Strangulation and stricture are very grave conditions.

Treatment.—In all acute forms of obstruction, food and cathartics should be withheld. Morphin and atropin, hypodermically, and warm applications to the abdomen will be required to relieve the pain. The stomach should be washed out two or three times daily, and in doubtful cases the colon may be irrigated with warm water. Distention of the colon with gas may be employed in suspected intussusception. Nutrient enemas may be resorted to to sustain the patient. An abdominal operation, performed early, is perhaps the best treatment in acute cases.

In chronic obstruction, efforts should be made to remove the fecal tumor. Large rectal injections of warm water or warm oil are very efficient. The administration of calomel, gr. ⅛ (0.008 Gm.), every hour is an additional aid. The rectal scoop may be employed if the obstruction is low down. Massage and electricity are often of value. Surgical intervention may be necessary.

INTESTINAL PARASITES.

TAPEWORMS.

Varieties.—*Tænia solium; Tænia saginata; Bothriocephalus latus; Tænia flavopunctata; Tænia confusa; Tænia echinococcus.*

Natural History.—In most cases the lower animals are the intermediary hosts by ingesting food infected by the tapeworm. The embryos or proscolices of these parasites are liberated in the stomach and from thence migrate to the muscles and various organs, becoming encapsulated, constituting scolices, or cysticerci. Meat infected by these encysted larvæ is said to be "measly." When ingested by man the capsules are dissolved and the scolices are liberated and become attached to the mucous membrane of the digestive tract developing into mature tapeworms.

Causes.—The *Tænia solium*, the "armed tapeworm," is one of the most common in this country. It is derived from the embryos contained in pork, known as the *Cysticercus cellulosae*.

The *Tænia saginata*, the "unarmed tapeworm," a not uncommon variety, is derived from the embryos contained in beef, known as *Cysticercus bovis*. According to the late Prof. Leidy, this is the form most frequently met with in this country.

The *Bothriocephalus latus,* also an "unarmed tapeworm," the largest parasite infesting man, is supposed to be derived from an embryo found in fish.

The *Tænia flavopunctata* develops its larvæ in lepidoptera and beetles. The intermediary host of the *Tænia confusa* is unknown.

The *Tænia echinococcus* occurs in its natural state in the intestines of the dog, the larval condition only, being encountered in man.

Faulty preparation, improper cooking, and lack of cleanliness as regards the food and drink are important etiologic factors.

Description.—The *Tænia solium* is from six to thirty feet in length, has a globular head, or scolex, a slender neck connecting its numerous flat segments or joints. The head, or scolex, measures about 1-40 of an inch, has a double circle of hooklets—whence the term "armed tapeworm"—and is provided with from two to four suckers. The segments or joints (*strobila*) are flat, and vary from one-eighth to one-half of an inch in length, and each contains both male and female sexual organs, the uterus being a long, numerously branched tube, in which the ova develop; the ova measure about 1-1700 of an inch in diameter. An ordinary tapeworm contains some five million ova. The parasite is firmly embedded in the mucous membrane of the upper third of the small intestines by its hooklets and suckers. The lower or terminal segments represent the adult and complete animal, and are termed the *proglottides,* which separate from the parasite and are discharged either alone or with the feces.

The *Tænia saginata* is from ten to forty feet in length, has a rounded or oval-shaped head, measures about 1-10 of an inch, and has four strong and prominent suckers, but no hooklets—whence the term "unarmed tapeworm;" the neck is short and thick and the segments are larger, stronger, and thicker than those of the tænia solium.

The *Bothriocephalus latus* is the largest of the three cestodes, the length ranging from fifteen to sixty feet, the head oval, measuring about 1-10 of an inch, a short neck, the segments or joints being nearly three times as broad as they are long. Its color is a dull, bluish-gray. Zoölogically considered, this variety is not a true

The *Tænia flavopunctata* is from 30 to 37.5 cm. long, and is nearly always encountered in young children. The joints are short and transversely linear. The anterior joints are immature, while posterior joints have a granular appearance due to immature ova. The most posterior joints are of a pale-brown color. A pair of longitudinal vessels traverse the joints and yellow spots, believed to represent the testicles, lie near the median line.

The *Tænia confusa* is about 5 cm. in length. Its terminal proglottides measure from 25 to 35 mm. long and 3.5 to 5 mm. wide. The scolex is 3 mm. wide and has four suckers and a retracted rostellum with several rows of hooklets.

The *Tænia echinococcus* is about 4 to 5 mm. long and is composed of three or four segments, of which only the end segment is mature. It is 2 mm. long and 0.6 mm. wide and contains about 5000 ova. The head is provided with a rostellum, two rows of hooklets, and four suckers. The adult worm is common to dogs in Iceland and Australia; human beings are affected only by the embryos, which, on being liberated in the digestive tract, migrate towards the periphery, particularly the liver, where they form hydatid cysts.

Symptoms.—Not infrequently there are no symptoms. In many cases, colicky pains, inordinate appetite, disorders of digestion, emaciation, anemia, constipation, cardiac palpitation, faintness, disorders of the special senses, choreic movements, convulsive seizures, and pruritus of the anus and nose are present more or less combined. The ingestion of a large meal often removes most of these symptoms. The presence of one or more segments of the tapeworm in the stools is conclusive.

Treatment.—The administration of an anthelmintic is necessary but should be preceded by restriction of the diet to liquids for one or two days with free purgation. Probably the best teniafuge is oleoresin of aspidium, fʒ ss (2 Cc.), alone or in combination.

℞.	Oleoresinæ aspidii, ...fʒ ij	8. Cc.
	Chloroformi, ...fʒ ij	8. Cc.
	Olei tiglii, ...♏ iv	.24 Cc.
	Glycerini, ...f℥ ij	60. Cc.

M. S.—Take half at 8 a. m.; the rest in an hour if needed (Dock).

Kousso, ℥i (30 Gm.), in a half-pint of water, fluid extract of the bark of the root of pomegranates, fʒ ss (2 Cc.), decoction of the bark of the pomegranate root (℥i (30 Gm.) to the pint (480 Cc.) of water) in divided doses, or the tannate of pelletierine, gr. x-xx (0.65-1.3 Gm.), may be used instead. A purgative should be also given after the anthelmintic and the stools carefully watched for the head of the parasite.

ROUND WORMS.

Varieties.—*Ascaris lumbricoides; Oxyuris vermicularis; Trichina spiralis; Anchylostomum duodenale; Filaria sanguinis hominis; Trichocephalus dispar.*

Causes.—The *Ascaris lumbricoides* is one of the most common of the parasites affecting the human family, and develops in the intestines, either after the entrance of the ova of the same, or from the so-called "intermediate parasites." Their entrance is effected by means of the food and drink.

The *Oxyuris vermicularis* develops in the large intestines, from either its peculiar ova or the so-called " intermediate parasite," these finding their way into the bowel with the food and drink, or by direct contact.

The *Trichinæ spiralis* are introduced into the human body by eating infected hog's flesh, either raw or but partly cooked.

The *Anchylostomum duodenale* gains access to the digestive tract by means of drinking water infected by the ova. It is common in the miners and brickmakers in Europe, Egypt, and India.

The *Filaria sanguinis hominis* is common in tropical countries, the medium of infection being contaminated drinking water. The parasites are found in the intestines of mosquitos, and on the sixth or seventh day of their development they change their habitat to water, through the death of the intermediary host.

The *Trichocephalus dispar* is one of the most common parasites in man, having for its habitat the cecum. The ova are very resistant to destructive agents and are found in large numbers in the feces. Infected drinking water is probably responsible for this parasite in man.

Description.—The *Ascaris lumbricoides,* or the round worm, is of a brownish color, having a cylindrical body, from ten to twenty tapeworm.

inches in length, and from an eighth to a fourth of an inch in circumference; the head terminates in three semilunar lips, each having about two hundred teeth. The ova are oval-shaped, are produced in immense numbers, some sixty-million in a mature female, have wonderful vitality, resisting extreme heat or cold. The round worm inhabits principally the small intestines, although it often migrates to other parts. They are found in numbers from one to several hundred.

The *Oxyuris vermicularis,* thread, or seat worm, resembles an ordinary piece of white thread, measuring from a sixth to a half inch in length, the head terminating in a mouth with three lips, the tail terminating as a sharp point. The ova are oval, produced in large numbers, each female containing about ten thousand, and are surrounded by a stout envelope, which increases their vitality. The seat worm, as its name indicates, inhabits the large intestines, especially the rectum, although they frequently migrate to the sexual organs. They vary in number, the parts frequented being entirely covered.

The *Trichina spiralis* presents itself in two forms; the intestinal trichina which is sexually mature and the muscle trichina which is sexually immature.

The *intestinal trichina* is a small, hair-like worm, the male measuring 1-18 of an inch, and the female 1-8 of an inch in length; the head is smaller than the rest of the body; the tail of the male has a bi-lobed prominence, between the divisions of which the anal opening is placed, and from which a single spiculum can be protruded; the female has a blunt, rounded tail, the reproductive outlet being situated toward the anterior part of the body; the ova are very small, containing embryos which are produced viviparously at the rate of at least one hundred each week after the entrance of the female into the intestinal canal.

The *muscle trichina* develops its sexual apparatus after it has entered the intestinal canal of the host. The viable embryos discharged from the female are in a state of motion, and at once migrate from the intestines to the muscular structure of the individual, and here set up inflammatory action, soon becoming surrounded by a capsule or shell in which they are coiled. After a time, in the muscle, the trichina undergoes a further change; lime salts being deposited in and about the capsule and in the parasite itself, when minute specks of lime are seen distributed throughout the muscular structure.

The development of the parasite from the period of impregnation up to time of sexual maturity is, under favorable conditions, less than three weeks. Within two days from the ingestion of the infected pork occurs the maturation of the muscle larvæ; in six days more the birth of embryos occur, and in about two weeks the migrating progeny have arrived at their habitat, the muscular structure.

The *Filaria sanguinis hominis* is an extremely small parasite and occurs in several forms. In the principal form (*Filaria Bancrofti*) the adult male is 83 mm. long and 0.4 mm. wide; the female is about 155 mm. long and 0.7 mm. wide. The ovum is 0.038 mm. long and 0.014 mm. wide. The adult worm is found usually in the lymphatics. The female produces an enormous number of embryos which migrate to various portions of the body and are found in the blood stream only during the sleeping hours, usually at night.

The *Trichocephalus dispar* is a delicate hair-like worm four to five centimeters long, the posterior two-fifths being the thickest portion. The terminal extremity of the female is conical and pointed, while that of the male is obtuse and round. The ova are 0.05 mm. long and oval in shape, provided with a small teat-like projection.

Symptoms.—The *Ascaris umbricoides*, or *round worm*, may be present in great numbers and yet produce no characteristic symptoms other than gastric and intestinal irritation, causing picking the nose, foul breath, colicky pains, nausea and vomiting, diarrhea, and disturbed sleep, such as tossing from side to side of bed and grinding the teeth. Any or all of these symptoms may be present or absent; a positive diagnosis is only possible upon the passage of the parasite.

The *Oxyuris vermicularis*, or seat worm, produces intense itching about the anus, with a desire for stool, the passages often containing much mucus, the result of the irritation produced by their presence. Should they migrate to the sexual organs, intense itching of these parts results, which, unless speedily corrected, leads in children to masturbation.

The group of phenomena produced by the *Trichina spiralis* is known as *trichinosis* and presents itself in three stages.

Intestinal stage, a gastrointestinal inflammation, with nausea, vomiting, and watery diarrhea, the severity depending upon the number of the parasites ingested.

Migration stage, a typhoid-life fever, rapid, feeble pulse, profuse sweats, intense thirst, dry tongue and lips, and red, swollen face, with soreness and tenderness of the muscular structure, increased by any muscular act. As a rule the mind is clear but decidedly apathetic.

Encapsulation Stage. If the number of parasites ingested has been few, recovery may occur in this stage; but if the number has been large, the gastroenteritis, fever, and muscular phenomena are severe, the patient is in a critical condition, between twenty and fifty percent succumbing.

The *Anchylostomum duodenale* is a blood-sucking parasite. It fastens itself to the mucous membrane of the digestive tract and at first produces colicky pains, diarrhea, and other symptoms of gastrointestinal irritation. The loss of blood induced by the parasite leads to anemia (Egyptian chlorosis, tunnel anemia), emaciation, and weakness.

The *Filaria sanguinis hominis* gives rise to a condition known as *filariasis* which is characterized by anemia, enlargement of the spleen, fever, chyluria, hematuria, lymphatic obstruction, and elephantiasis.

The *Trichocephalus dispar* may be unattended by symptoms, or may be accompanied by slight gastrointestinal irritation, anemia, cerebral manifestations, and in rare instances beri-beri.

Treatment.—The *Ascaris lumbricoides* is readily removed by the following "worm powder":

℞.	Santonini	gr. ¼-j-ij	.016-.065-.13 Gm.
	Hydrargyri chlor. mitis.	gr. ⅓-ij	.022-.13 Gm.

M.—Ft. chart.

S.—At bedtime, followed by a dose of castor oil before breakfast.

For the *Oxyuris vermicularis* the above santonin powder, with the use of enemas of quassia, alum, salt or

℞.	Acidi carbolici	gr. v-x	.3-.6 Gm.
	Glycerin	♏v-x	.3-.6 Cc.
	Aquæ	Oj	480. Cc.

M. S.—For rectal injection.

An enema of corrosive sublimate (1 to 10,000) is sometimes employed. The rectal injection should not be retained and should always be preceded by a large enema of water to thoroughly cleanse the bowel. Washing of the anus and external genitals with a carbolic acid solution is often useful and aids in allaying the intense itching.

In *trichinosis,* the pork should be so prepared as to kill any existing trichinæ. If the patient is seen within four or five days after the ingestion of the parasites, emetics, purgatives, lavage, and intestinal irrigation are indicated. Following these procedures vermicides may be administered, such as glycerin (one part) and water (two parts), ʒi or 4 Cc. every hour, benzine, ʒi (4 Gm.), in capsules, quinin, and santonin. The muscular pains may be relieved by hot applications and morphin hypodermically. Tonics and stimulants are necessary to sustain the patient. The mortality ranges from five to thirty percent.

Anchylostomiasis requires thorough boiling of the drinking water in districts known to be infected. Thymol, gr. xxx (2 Gm.), in divided doses followed by a purgative, seems to have a specific action. The anemia calls for rest, iron, quinin, strychnin, arsenic, etc.

Filariasis is most difficult to treat. The symptoms should be met as they arise. Thymol and methylene blue may be tried.

Trichocephalus dispar has no special treatment.

DISEASES OF THE PANCREAS.

ACUTE PANCREATITIS.

Definition.—An acute inflammation of the pancreas affecting, primarily, the fibrous and fatty interstitial tissue.

Causes.—It is most common in males after forty-five years of age and may result from gastrointestinal disorders, impaction of gall-stones, traumatism, infectious fevers, and local bacterial infection.

Pathologic Anatomy.—The appearances of the organ differ somewhat according as to whether the termination is hemorrhagic, gangrenous, or suppurative.

In the *hemorrhagic form* the organ is enlarged and infiltrated with blood in various stages of decomposition, and scattered between these hemorrhagic foci are white areas of fat-necrosis. Round cells and red blood corpuscles are found in the ducts and acini and bacteria are present in large numbers. The extravasation of blood may be extensive, involving the adjacent tissue and the various peritoneal folds.

The *gangrenous form* is a later stage of the preceding. The tip, or even the entire organ, is converted into an offensive, soft, slate-colored mass. Partial or complete sequestration of the gland, in the small omental cavity, may result, often its only attachment being a few threads. Peritonitis accompanies it, and fat-necrosis may be present. Thrombosis of the splenic and portal veins may occur.

The *suppurative form* is also a terminal affection. The pancreas is enlarged and the seat of numerous small abscesses and intervening hyperemic areas. Diffuse suppuration may occur, or in chronic cases a solitary abscess may be formed. Localized peritonitis is present in many cases. Thrombosis of splenic and portal veins may also occur.

Symptoms.—The onset is sudden with intense abdominal pain and tenderness in the epigastrium, and vomiting. The upper left quadrant of the abdomen becomes distended and tympanitic, and the temperature is slightly above or below normal. The symptoms of collapse soon present themselves, and the patient succumbs, as a rule, within three days. The occurrence of chills, fever, marked abdominal distention, tenderness, and tympany, and jaundice, in addition indicates a termination by gangrene. Suppuration is attended by irregular fever, jaundice, constipation, and prolongation of life for three or four weeks.

Diagnosis.—Intestinal obstruction, perforation of the stomach, acute toxic gastritis, and biliary colic resemble this affection in many of its symptoms, and the clinical history must be relied upon to a great extent in distinguishing them.

Prognosis.—The disease is almost always fatal.

Treatment.—Surgical intervention offers the only hope, as medicinal measures are useless.

CHRONIC PANCREATITIS.

Definition.—A condition in which there is interstitial overgrowth of the pancreas, increasing the density and size of that organ and compressing the secreting structure. Pigmentary deposits may be present, and calculi may be lodged in the ducts.

Causes.—In rare instances it follows the acute form but more frequently is due to arterial sclerosis, alcoholism, syphilis, obstruction of the pancreatic duct, extension from gastrointestinal inflammation, or diabetes.

Symptoms.—There are no characteristic symptoms. Paroxysmal pain, abdominal distention, indigestion, diarrhea, fatty stools, jaundice, albuminuria, and glycosuria may be present in varying combinations.

Treatment.—There is no special treatment unless impacted gall-stones are detected as a cause, when surgical procedures will be found very valuable. The elimination of fats and starches from the diet is advised. The treatment suggested for diabetes mellitus is applicable to this condition. The course is very slow, and with the appearance of glycosuria, the outlook becomes proportionately grave.

CANCER OF THE PANCREAS.

Pancreatic cancer is a rare condition. The growth, as a rule, is primary, and of the scirrhous variety, affecting first the head of the organ. It is most common in males past forty years of age.

Symptoms.—The most important symptoms are the tumor in the region of the pancreas, jaundice, and fatty or greasy stools. Associated with these are usually dull epigastric pain, indigestion, weakness, emaciation, anemia, and cachexia. As the tumor enlarges and emaciation progresses, the aortic pulsation may be transmitted to it. Ascites may result from pressure on the portal vein. Diabetes mellitus may also occur.

Diagnosis.—The location of the tumor, jaundice, and fatty stools will aid greatly in distinguishing this affection from pyloric cancer, with which it may sometimes be confused.

Treatment.—The treatment is symptomatic and very unsatisfactory, as all cases terminate fatally.

CYSTS OF THE PANCREAS.

Cystic tumors of the pancreas are, as a rule, retention cysts due to the closure of the duct of Wirsung, by concretions, tumors, or cicatrices, but they may result from encapsulation of extravasated blood, echinococcus disease, or malignant tumors. Congenital cysts are sometimes encountered.

Symptoms.—If the cyst is of sufficient size to induce suspension of pancreatic function, fatty diarrhea will usually be present. An enlargement may be usually made out in the left portion of the epigastrium, between the costal cartilages and the median line, which will be globular, resisting, and inelastic. Aspiration of the tumor will yield a fluid of brown or chocolate color, which is capable of emulsifying fats and converting starch into sugar. Abdominal pain, digestive disturbances, and emaciation accompany it.

Treatment.—Exploratory incision is indicated after withdrawal of the characteristic fluid. The outlook is guardedly favorable.

PANCREATIC CALCULI.

Calculi in the pancreas may be regarded as inspissated particles of altered pancreatic secretion, around which concretions of carbonate and phosphate of lime occur. They are multiple and about the size of a pea, being found in the pancreatic duct and its branches.

Symptoms.—They may be unattended by symptoms or there may be pancreatic colic, glycosuria, fatty stools, and the passage of calculi by the bowel.

Treatment.—Morphin and atropin are required to relieve the pancreatic secretion.

DISEASES OF THE PERITONEUM.

PERITONITIS.

Synonym.—Inflammation of the peritoneum.

Definition.—A fibrinous inflammation of the peritoneum, either acute or chronic, characterized by fever, intense pain, tenderness, tympanites, vomiting, and prostration. It may be limited to a part, *local,* or it may involve the entire membrane, *general,* peritonitis.

Causes.—The *acute variety* arises from bacterial infection and may follow exposure to intense cold, protracted irritation of the abdomen by blisters, traumatism, penetrating wounds of the abdomen, inflammation or perforation of the stomach, intestines, gall or urinary bladder, vermiform appendix, or the surrounding parts, inflammation of the pelvic viscera, pyemia, septicemia, erysipelas, hernia, pleurisy, articular rheumatism, and nephritis.

The *chronic variety* may succeed an acute attack, but is more commonly due to tuberculosis, cancer, nephritis, or syphilis.

Pathologic Anatomy.—In the *acute form,* the membrane is hyperemic, and there may be scattered extravasations of blood from rupture of the distended capillaries. The secretion is arrested and the peritoneum becomes dry, lusterless, and opaque. The inflammatory exudate may be serofibrinous, fibrinous, or purulent. A serous or serofibrinous exudation is productive of more or less ascites. As the fluid is absorbed the fibrinous portions remain behind and become organized, giving rise to adhesions between opposing surfaces. If the inflammation subsides in the early stage, or if the fibrin is in excess from the beginning, adhesions form without ascites. These adhesions may serve to wall off any localized purulent exudation, thereby converting it into an abscess. The inflammatory process may be diffused or circumscribed.

In the *chronic form,* the peritoneum is thickened and studded with tuberculous or cancerous nodules, as the case may be. Adhesions are present and serve to mat the intestines together, and to disturb the relations of the abdominal viscera. There is a varying quantity of fluid in the cavity, which is albuminous, but may, at times, be bloody. The omentum is greatly thickened and shrunken.

Symptoms.—*Acute peritonitis* is manifested by a sudden onset with a chill, fever, 101°-103° F., and a tense and wiry pulse, 100-140. There is present also intense abdominal pain and tenderness. The patient lies motionless on the back, with the legs and thighs flexed. The expression is anxious, and the excruciating cutting or boring pain causes the features to appear pinched. The abdomen is distended and rigid from constipation, effusion, and meteorism. The breathing is shallow and thoracic, and the diaphragm is more or less fixed, and in severe cases is pushed up as far as the third or fourth rib, causing compression of the lungs and displacement of the heart, liver, and spleen. Impairment of appetite, intense thirst, nausea, vomiting, and hiccough are present. In the early stage, when the abdomen is distended with gas, percussion will yield a tympanitic note, but later, as the exudate is poured out, dullness will be obtained, corresponding to the situation of the exudate; small and fixed if circumscribed, but if the exudate is large and diffuse, the dullness will be movable. The course of the disease is rapid, especially in severe cases, collapse supervening, being indicated by subnormal temperature or a rapid decline in the existing fever, cold, clammy skin, rapid, feeble pulse, weakness, and the Hippocratic expression (sunken eyes, collapsed cheeks and temples, pinched nose, and drawn upper lip). When the condition is produced by extension, it begins with local and gradually increasing pain, tenderness, and rigidity, rising temperature, tense pulse, and vomiting. When it follows perforation it is ushered in with severe pain and all the symptoms of shock. In purulent peritonitis, hectic phenomena are present In ordinary cases, peritonitis runs its course from six to eight days, terminating in collapse or a protracted convalescence.

Chronic peritonitis is usually tubercular in origin, and is attended by irregular chills, fever, and sweats, distended abdomen, constipation, alternating with diarrhea, diffused tenderness with points of intensity and hardness, colicky pains during digestion, rapid emaciation, anemia, and loss of strength. Usually the lower portions of the abdomen yield a dull note on percussion from the presence of fluid, or scattered but fixed points of dullness showing the presence of encysted fluid. Palpation may detect a friction fremitus, and sometimes the nodules may be felt. When the fluid exudate is of considerable quantity fluctuation may be elicited.

Diagnosis.—The characteristics of acute peritonitis are the sudden onset, intense abdominal pain, tenderness, rigidity, and distention, the decubitus, the quick wiry pulse, the fever, the constipation, the short course, and the Hippocratic expression. These are present to a less degree in chronic peritonitis, which is most readily distinguished by the history and the results of physical examination.

Acute gastritis differs from peritonitis in having a history of corrosive poisoning or dietetic indiscretion, early and severe vomiting, severe pain limited to the stomach, diarrhea at times, and no marked abdominal distention, tenderness, or rigidity.

Acute enteritis has localized pain and tenderness, and diarrhea is almost invariably present.

Rheumatism of the abdominal muscles is subacute; rigidity, pain, and tenderness are present, but abdominal distention, constipation, and marked constitutional symptoms are absent.

Biliary colic is attended by pain, localized in most cases to the hepatic area; rigidity is present to some extent; and jaundice is common. The pain, tenderness, rigidity, and distention, as seen in peritonitis, are absent.

Renal colic is characterized by paroxysmal pain, which begins posteriorly and follows the course of the ureters, and is attended by altered urinary secretion and retraction of the testicle.

Typhoid fever when attended by marked distention and tenderness may resemble peritonitis, but the history, temperature record, Widal test, rose-colored eruption, and diarrhea in the former affection will distinguish them. The occurrence of peritonitis, secondary to perforation, in the course of typhoid fever, is usually announced by a fall of temperature, marked tympanites, sudden localized pain, quick wiry pulse, and collapse.

Intestinal obstruction is marked by absolute constipation and stercoraceous vomiting and less abdominal pain and tenderness. Rupture of an obstructed bowel is succeeded by peritonitis.

Hysterical abdomen may be confused with peritonitis, but its occurrence in neurotic women, its tendency to recur, and the absence of fever and the characteristic pulse will aid materially in making the proper diagnosis.

Circumscribed peritonitis is difficult to detect and requires for its recognition a careful history and physical examination, and often the use of the exploring needle.

Prognosis.—In acute peritonitis of septic origin the termination is fatal usually within a week. In perforative cases following typhoid fever, Keen has shown that from 15 to 30 percent recover with surgical treatment within 24 hours. Localized peritonitis is more favorable; an abscess may be formed which may rupture spontaneously or be evacuated at a later period by a surgeon.

In chronic peritonitis the prognosis is unfavorable, but the duration is considerably longer than the acute form. Tuberculous peritonitis, when properly treated, may end in recovery or at least suspension of the disease-process for a considerable period.

Treatment.—The surgeon should be consulted very early in the *acute variety*, as operation offers the only hope in many cases, especially those due to perforation. Counterirritation may be employed, but is often useless. Morphin and atropin, hypodermically, will relieve pain, control vomiting, and lessen peristalsis. If the stomach is retentive, ice, milk, and lime-water, champagne, and brandy, may be given. Turpentine stupes and turpentine enemas are frequently beneficial. Belladonna and mercury ointment applied locally is of value at times. Strychnin, quinin, and other stimulants may be necessary. In non-perforative cases, saline purgatives may be employed in concentrated solutions, administering one or two drams of Rochelle or Epsom salt every two hours until there is free bowel movement. In circumscribed forms, leeches, blisters, and hot applications may produce considerable relief. During convalescence, rest in bed, nutritious diet, moderate stimulation, and the following are indicated:

℞.	Potassii iodidi ....gr. v-x	.3-.6	Gm.
	Ferri pyrophos. ....gr. ij	.13	Gm.
	Elix. simpl. ....fʒ ss	2.	Cc.
	Aquæ destillatæ.....ad fʒ ij	ad 8.	Cc.

M. S.—To be given every six hours.

Chronic peritonitis will require the application of tincture of iodin to the abdomen, rest in bed, and the administration of opium, potassium iodid, cod-liver oil, and stimulants. Surgical treatment is necessary in many cases.

ASCITES.

Synonyms.—Peritoneal dropsy; hydroperitoneum.

Definition.—A collection of serous fluid in the abdomen, or more correctly in the peritoneal cavity; characterized by a distended abdomen, fluctuation, dullness on percussion, displacement of viscera, embarrassed respiration, plus the symptoms of its cause. The quantity of fluid in the peritoneal sac varies from a few ounces to many gallons. It is generally of a straw color, or at times greenish, and is transparent, and has an alkaline reaction. The specific gravity is from 1010 to 1020. When blood is present in any great quantity, it points to cancer as a cause. The peritoneum becomes cloudy, sodden, and thickened, from long contact with the fluid.

Causes.—It may be a part of a general dropsy such as follows chronic heart, lung, or kidney disease, or it may be due to chronic peritonitis or mechanical obstruction of the portal circulation from hepatic cirrhosis, portal thrombosis, or abdominal tumors. It may accompany intense anemia.

Symptoms.—The onset is gradual and considerable swelling of the abdomen may occur before the disease attracts attention. The umbilicus is forced outward and constipation, scanty urination, and embarrassed respiration and cardiac action result from the pressure of the accumulated fluid.

Physical examination reveals on *inspection,* distention of the abdomen, the surface of which is smooth and shining; broadening of the base of the thorax; bulging of the flanks when the dorsal position is assumed; and enlargement of the superficial veins. On *palpation* a peculiar wave-like impulse is imparted to the hand lying on the side of the abdomen, while gently tapping the opposite side. On *percussion* dullness will be obtained over the fluid, which always seeks dependent parts, above which a tympanitic note will be heard. The dullness is movable, changing its position with the changes in the patient's posture.

Diagnosis.—*Ovarian tumors* differ from ascites in their history, unilateral enlargement limited as a rule to either iliac region, and fixed dullness, which is usually anterior. Combined vaginal and abdominal, or rectal examination will serve to detect the tumor.

Tympanites is characterized by enormous distention of the abdomen, but percussion yields universal tympany.

Pregnancy differs from ascites in its history, shape of the abdomen, fixed dullness, and the concomitant signs, cessation of the menses, enlargement of the breasts, fetal heart-sounds, changes in uterus and vagina, etc.

Distention of the bladder may be distinguished from ascites by the location of fixed dullness, the presence of tenderness over the bladder, the history of suppression of urine, and by the relief following catheterization.

Chronic peritonitis may be differentiated by its history, pain, tenderness, more or less vomiting, thickened abdominal walls, smaller effusion with less range of motion due to adhesions, smaller area of dullness, and its common association with tuberculosis or cancer.

Prognosis.—In the common form due to organic disease, the prognosis is unfavorable, for while the dropsy may be removed, it rapidly returns. In peritoneal cases it is more favorable, and in idiopathic cases, which are rare, it terminates in health within a few weeks.

Treatment.—The first indication is to treat the cause of the ascites and the second to remove the fluid.

Three modes of removing the fluid present themselves: *first,* by hydragogue cathartics; *second,* diuretics and diaphoretics, and *third,* tapping. The first and second modes may be combined, as follows:

℞. Pulv. jalapæ comp......ʒ j-ij   4.-8. Gm.
S.—In water, an hour before breakfast;

And—

℞. Potassii acetat. ....gr. xxx   2. Gm.
Spts. ætheris nitrosi ....♏xv   1. Cc.
Infus. digitalis..q. s. ad fʒ ij   ad 8. Cc.
M. S.—Every six hours.

Or, instead use the following:

℞. Hydrargyri chlor. mitis..gr. iij   .2 Gm.
Ext. opii ....gr. 1-12   .005 Gm.
M. S.—One every three or four hours.

If these fail, as they certainly will after a time, the embarrassed respiration and cardiac action will call for tapping, which may be performed with the trocar or the aspirator. The tapping does not remove the cause, and the fluid often rapidly accumulates again. In performing this operation the patient should be placed in a semirecumbent posture, and the area selected (usually in the median line between the umbilicus and the symphisis) should be anesthetized by ethyl chlorid or a mixture of ice and salt. After the aspirator has been thrust into the abdomen, the trocar portion is withdrawn and the liquid flows through the cannula. When the liquid has been entirely removed the instrument is withdrawn, the opening sealed, and a rather tight abdominal binder, with a pad of cotton or gauze applied. Collapse should be watched for and carefully guarded against during this procedure. Before tapping always examine the bladder, using the catheter if there be any doubt.

DISEASES OF THE BILIARY PASSAGES.

CATARRHAL JAUNDICE.

Synonyms.—Catarrh of the bile-ducts; icterus.

Definition.—An acute catarrhal inflammation of the mucous membrane of the bile-ducts and of the duodenum; characterized by gastrointestinal derangement, yellowness of the skin and sclera, itching of the skin, feverishness, and mental depression.

Causes.—Extension of gastrointestinal inflammation such as follows a debauch, excesses in eating and drinking, and exposure is the most common cause. Atmospheric changes, passive congestion of the liver, and the infectious fevers, such as pneumonia, malaria, relapsing fever, etc., are less frequent factors in its production.

Pathologic Anatomy.—The mucous membrane of one or more of the bile-ducts, or of the duodenum, becomes hyperemic, swollen, and thickened, from an effusion of serum into the submucous tissue; the result of this condition is the closure of the biliary passages, thereby impeding the outward flow of bile. The bile in the hepatic ducts being retained by the obstruction, results in a staining of the liver substance and an absorption of bile, with its appearance in the blood.

Symptoms.—The affection begins with epigastric distress, coated tongue, impaired appetite, nausea, with perhaps vomiting, and looseness of the bowels and slight feverishness. In from three to five pain. Pilocarpin has been advised for its stimulant effect on days the eyes become yellow and jaundice gradually appears over the whole body; the feverishness disappears, the skin becomes harsh, dry, and itchy, the bowels constipated, the stools whitish or clay-colored, accompanied with much flatus, and colicky pains; the urine heavy and dark, loaded with urates and containing biliary elements. A few drops of the urine placed on a whitish surface, and a drop or two of nitric acid made to flow against it, will exhibit the following "play of colors:" a greenish tint, from the conversion of bilirubin into biliverdin, quickly followed by blue, violet, red, and yellow, or brown. When the jaundice is complete, the surface is cold, the heart's action slow, the mind torpid and greatly depressed, and pain or tenderness on pressure over the hepatic region.

The symptoms subside within a few days after the jaundice appears, but the depression, discoloration, and condition of the bowels persist for one or two weeks.

Diagnosis.—*Catarrhal jaundice* may be recognized by its acute course, the history, the mild symptoms, the age, and the termination. Jaundice is readily detected by examination of the mucous membranes of the eyes and mouth, the clay-colored stools, and by the reaction of the urine to nitric acid. The discoloration of the skin alone is not positive.

When jaundice is induced by obstruction to the outflow of bile other than that produced by inflammation, such as arises from stricture of the common duct, tumors of the abdominal viscera, foreign bodies such as gall-stones and parasites, fecal accumulations, spasm of the bile ducts due to emotion, etc., the symptoms of these different affections will be found associated with the icteroid manifestations.

Icterus neonatorum is the variety that occurs in children, and may be due to a patulous ductus venosus which allows the portal blood, rich in bile, to enter the circulation, or to some morbid condition of the liver or bile-duct which causes insuperable obstruction. In the first variety, recovery is the rule within a few days, to a week or more, but in the second the termination is usually fatal.

Non-obstructive or hematogenous jaundice is unassociated with inflammatory changes in the bile-ducts, and arises from disintegration of the blood or hemolysis. It may be caused by poisons, such as nitrobenzol, chlorates, snake-venom, chloroform, phosphorous, etc., yellow fever, relapsing fever, bilious fever, pernicious anemia, pyemia, typhoid fever, acute yellow atrophy of the liver, and similar conditions. It differs from catarrhal jaundice in its history, the absence of clay-colored stools, and less staining of the urine.

Treatment.—The patient should be placed at rest in bed and the diet restricted to milk and lime-water, broths, eggs, lean meats, etc., being careful to eliminate all starchy, fatty, or saccharine substances. Calomel, gr. ¼ (0.016 Gm.), with sodium bicarbonate, gr. iii (0.2 Gm.), should be then given every two hours until twelve doses are taken, followed by Hunyadi water or the following:

℞.	Sodii bicarb. ....ʒ iv	15. Gm.
	Tinct. nucis vom.......fʒ iv	15. Cc.
	Tinct. capsici ....fʒ j	4. Cc.
	Tinct. rhei ....f℥ ij	60. Cc.
	Inf. gent. comp..q. s. ad f℥ vj	ad 180. Cc.

M. S.—Dessertspoonful every four or five hours, in water.

Sodium phosphate, ʒi (4 Gm.), may also be given, well-diluted, every four hours. The dry, itching skin, may be relieved by diaphoresis, a hot bath, containing potassium carbonate, night and morning, or a weak carbolic acid solution. If insomnia is present potassum bromid, gr. xxx (2 Gm.), may be administered. Diuretics are indicated if the urine continues scanty, preference being given to the alkaline waters, potassium bitartrate lemonade, and spirits of nitrous ether, ♏x-xx (0.6-1.3 Cc.). In cases in which the constipation persists, aloes, podophyllum, colocynth, and other cholagogues should be employed. Irrigation of the colon once daily with cold water, gradually increasing the temperature, is often very effective. During convalescence, and when the condition tends to become chronic, the following is of great benefit:

℞.	Strychninæ sulph. ......gr. ss	.03 Gm.
	Acid. nitrohydrochloric. dil. ....fʒ iv	15. Cc.
	Tinct. gentian. comp....f℥ iiss	75. Cc.

M. S.—Teaspoonful after meals, well-diluted.

BILIARY CALCULI.

Synonyms.—Hepatic calculi; gall-stones; hepatic colic; cholelithiasis.

Definition.—Concretions originating in the gall-bladder, or biliary ducts, derived partly or entirely from the constituents of bile. Their presence is generally unrecognized until one or more attempt to pass along the ducts, when an attack of hepatic colic is produced.

Causes.—Gall-stones result from the precipitation of the crystallizable cholesterin and its combination with inspissated mucus in the gall-bladder or ducts. Bacteria are causal factors in that they induce inflammatory changes in the gall-bladder in consequence of which cholesterin and lime salts are excreted in excess and deposited. The affection is most common in women past middle life, particularly in those who have abdominal tumors or have borne a number of children. Obesity, sedentary habits, excesses in eating and drinking, tight lacing, typhoid fever, and malignant disease of the stomach and liver are also etiologic factors.

Pathology.—Biliary calculi vary greatly in size and number. As a rule, the stone is brown and spherical, oval, or polygonal. The shape varies according to the manner in which the calculi are packed together during their formation. Cholesterin is the chief constituent, but bile-pigment and lime salts may also be present. On section, the calculus shows the manner of its formation by the concentrically arranged layers of different color. Commonly several stones exist, and they are generally found in the gall-bladder or cystic duct, but may very rarely be found in the liver or hepatic duct.

Symptoms.—The manifestations of biliary calculi vary according to the course of the affection. While they remain quiescent in the gall-bladder they may occasion but very little discomfort and often remain undetected for a long period. Often they induce expulsive efforts of the gall-bladder by their irritation, and may be pushed on into the bowel, into the cystic duct, or into the common duct. This event is always marked by hepatic or biliary colic. If the calculi pass completely into the bowel and are not too large, they may appear in the stools, more often they slip back into the gall-bladder and the attack subsides to recur at a later period. If they pass into the cystic or common duct, and neither find a free exit nor slip back into the gall-bladder, impaction, perforation, peritonitis, suppurative cholecystitis, suppurative angiocholitis, and hepatic abscess are the possible terminations. Irritation of the gall-bladder by gall-stones is believed by some observers to ultimately induce malignant disease of the biliary passages.

Hepatic colic commences suddenly at the moment a calculus passes from the gall-bladder into the cystic duct with piercing, agonizing pain, which begins over the gall-bladder and spreads over the abdomen to the chest and right shoulder. Tenderness and rigidity are present over the gall-bladder and also extend over the abdomen. Nausea, vomiting, a small feeble pulse, cool skin, pale distorted anxious expression, fainting, spasmodic trembling, chills, moderate fever, and sometimes convulsions, accompany the attack. The paroxysm lasts from an hour to two or more days, with remissions until the calculus reaches the duodenum, when the pain suddenly ceases. When the stone is obstructed in its passage jaundice results.

Obstruction of the cystic duct by an impacted gall-stone may be followed by very few symptoms, and jaundice is absent. It may give rise to dropsy or atrophy of the gall-bladder or cholecystitis.

Obstruction of the common duct by an impacted calculus is characterized by persistent jaundice, paroxysmal pain, and ague-like attacks of chills, intermittent fever, and sweats. Nausea and vomiting may be present, and there may be enlargement of the liver and spleen. The stools are sometimes bile-stained. These symptoms may continue for months or years, and if the obstruction is not relieved, suppurative cholangitis, perforation, or fibroid induration, dropsy, or atrophy of the gall-bladder may result.

Diagnosis.—*Hepatic colic* may be mistaken for *renal colic* but in the latter affection the pain begins in the lumbar region and follows the line of the ureters into the genitals. The urine is bloody and may contain the stone. Jaundice is absent.

Intestinal colic is attended by diffuse abdominal pain and distension. Flatulence is present and on its discharge the pain is relieved.

Pleurisy on the right side may cause some confusion, but the friction sound and the limitation of breathing with sharp pain on inspiration will aid in making a distinction.

Appendicitis is characterized in most cases by pain, tenderness, and rigidity in the right iliac region, and by the absence of jaundice and bile-stained urine.

Gastralgia is usually attended by paroxysmal pain over the region of the stomach, which is relieved by pressure and by taking of food.

Gastric ulcer may be accompanied by paroxysmal pain, under which circumstances it is usually induced by eating, and in addition localized epigastric tenderness, hematemesis, and hyperacidity are present.

Pseudo-biliary colic may resemble this affection very closely, but its occurrence in neurotic women, and the absence of calculi in the stools, should be remembered in making a diagnosis.

Prognosis.—Uncomplicated cases terminate in recovery as a rule. The occurrence of ulceration, suppuration, or perforation, is of grave significance.

Treatment.—During an attack of hepatic colic, morphin and atropin should be administered hypodermically, and warm fomentations applied over the region of the liver and gall-bladder. Chloroform may be necessary in some cases to relieve the pain. If there is any tendency towards collapse, a hot bath and diffusible stimulants should be administered.

Succeeding the attack and during the intervals, the diet should be carefully regulated, eliminating all fatty and saccharine substances, and the patient should be instructed to avoid all excesses and indulge moderately in exercise. Water-drinking should be encouraged, and when possible the saline mineral waters, such as Carlsbad, Vichy, and Saratoga waters should be employed. Sodium bicarbonate or sodium phosphate, ʒi (4 Gm.), may be administered well-diluted before meals. When constipation tends to exist, Rochelle or Epsom salt should be given regularly to overcome it. Ether, turpentine, and sweet oil have been recommended as solvents, but their efficiency in this respect is doubtful. The succinate of sodium, gr. v (0.32 Gm.), administered three times daily is accredited with beneficial properties in preventing recurrences.

Surgical intervention is indicated when complications threaten and when the symptoms are so intense and persistent as to endanger the patient's vitality.

ACUTE INFECTIOUS CHOLECYSTITIS.

Definition.—Acute inflammation of the gall-bladder.

Causes.—The affection is always due to infection by pathogenic bacteria, especially the colon bacillus and typhoid bacillus. Occasionally the pneumococcus, staphylococcus, and streptococcus are the exciting causes. It may follow irritation from gall-stones, pneumonia, or typhoid fever.

Pathologic Anatomy.—The inflammation may be of varying grades. In mild forms the exudate is mucoid or muco-purulent; in the more severe forms it is purulent and the inflammation may proceed to ulceration, perforation, or gangrene. In rare instances the gall-bladder may be distended with blood.

Symptoms.—Pain at the border of the thorax to the right of the median line is invariably present, and the gall-bladder is enlarged and tender. Vomiting is common. Fever accompanies the condition and jaundice may or may not be present. In the presence of pus the fever becomes irregular and attended by chills and sweats, and an examination of the blood will show leukocytosis.

Diagnosis.—The features of this affection that serve to distinguish it are the history, the preceding affection and the location of the pain and circumscribed tenderness. The presence of a tumor in the region of the gall-bladder is confirmatory.

Prognosis.—Many mild catarrhal cases undoubtedly terminate in recovery without being recognized. Suppurative cases are unfavorable and tend towards a fatal termination. Prompt surgical intervention offers the only hope.

Treatment.—The symptoms should be treated as they arise on general therapeutic principles. The case should be placed in the care of a skilled surgeon as soon as detected.

DISEASES OF THE LIVER.

Preliminary Considerations.—Normally, the greater portion of the liver is situated in the right upper quadrant of the abdomen, a small portion extending over the median line into the left upper quadrant. Percussion over the area occupied by the liver yields a dull note. Absolute liver dullness extends in the median line from the lower border of cardiac dullness to midway between the ensiform appendix and the umbilicus; in the mammary line from the upper border of the sixth rib to the costal margin; in the axillary line from the eighth to the eleventh rib; and posteriorly from the tenth to the eleventh rib.

The situation of the liver may be altered as the result of transposition of the viscera, tight-lacing, ascites, abdominal tumors, pleurisy, or emphysema. Varying degrees of displacement may be encountered. Floating liver is a rare condition of this kind, in which relaxation and elongation of its ligaments permit it to fall from its normal position, especially when the erect posture is assumed. Tight lacing and pendulous abdomen are given as causes. The organ is recognized in its new position by its shape and dullness and its absence from the normal situation aids in confirming the diagnosis.

The principal abnormality in shape of the liver is that known as the "laced-off" liver which results from the wearing of tight waist bands and corsets. It is characterized by division of the right lobe into two almost equal parts by a transverse furrow. The connection between the two parts is in very rare cases reduced to a fibrous band. The affection is most common in women and seems to favor cholelithiasis. There are no symptoms, as a rule, and the possibility of this condition should be considered in examining for floating kidney, and other visceral displacements.

In the physical examination of the liver it should be remembered that normally the edge of the liver is seldom felt by the examining fingers except in thin and emaciated subjects and in young children. It becomes palpable when enlarged or displaced from any cause. Irregularities of the surface of the liver suggest the possibility of cancer, syphilis, abscesses, or hydatids. Alterations in the consistency of the hepatic structure are indicative of certain changes, thus in cancer, congestion, hypertrophic cirrhosis, and amyloid infiltration, the liver is more dense while in abscesses and hydatid cysts the consistency is less, particularly in the diseased area. If in the examination, tenderness is elicited it points to the presence of congestion, inflammation, abscess formation, or cancer. Pulsation is occasionally encountered and is nearly always due to passive congestion following tricuspid regurgitation, but it may also be due to aneurysm or a tumor in close proximity to the abdominal aorta which transmits its pulsation.

The size of the liver may give an indication of the character of the affection in many instances, thus hypertrophic cirrhosis, congestion, cancer when diffuse, and fatty and amyloid conditions produce a more or less uniform enlargement of the organ; in cancer, gumma, abscess, hydatid cysts, and similar affections the enlargement is somewhat nodular in character; and in atrophic and degenerative conditions the organ undergoes diminution in size. An apparent enlargement may occur when the liver dullness is increased by any pulmonary condition that displaces the organ downwards, and an apparent diminution may be observed when the liver dullness is obscured by tympanites from any cause, or by pulmonary or subcutaneous emphysema.

CONGESTION OF THE LIVER.

Synonyms.—Torpid liver; biliousness.

Definition.—An abnormal fullness of the vessels of the liver, with consequent enlargement of that organ; it is termed *active* when arterial; *passive* when venous. The condition is characterized by torpidity of the digestive and mental functions, and slight jaundice.

Causes.—*Active Congestion;* heat, atmospheric or artificial; habitual constipation; malaria; excesses in eating and drinking; alcoholic or malt liquors. In females, an arrested menstrual epoch may give rise to an attack.

Passive congestion; cardiac and pulmonary diseases.

Pathologic Anatomy.—The liver is enlarged in all directions, and is abnormally full of blood. Cases due to obstructive diseases of the heart or lungs present the so-called "nutmeg liver" appearance. At the center of each lobule the dilated radicle of the hepatic vein, enlarged and congested, may be discerned, while the neighboring parts of the lobule are pale, the radicles of the portal vein containing less blood. Long-continued congestion establishes atrophic degeneration or cyanotic induration of the organ; the decrease in size is confounded with the condition of cirrhosis, but the "atrophic liver" is smooth, while the "cirrhotic liver" is nodulated.

Symptoms.—*Acute congestion* begins with malaise, aching of the limbs, feverishness, headache, depression of spirits, coated tongue, anorexia, nausea, and sometimes vomiting. Constipation and flatulence are present, and there is a feeling of fullness, weight, and soreness in the hepatic region with dull pain extending to the right shoulder. The liver is uniformly enlarged and tender. The complexion is muddy and there may be slight jaundice. The attack usually lasts about a week.

Passive congestion is characterized by similar symptoms but of less severity. The onset is gradual and gastrointestinal catarrh is common. In addition, there are the symptoms of the causal heart or lung disease.

Prognosis.—The acute attacks end favorably but if there is a constant repetition of them, atrophic degeneration is the usual result. Passive congestion is dependent entirely upon the severity of its cause for its prognosis. In many cases atrophic degeneration or cyanotic induration follows.

Treatment.—In acute attacks induced by dietetic indiscretions the following mixture should be given:

℞. Sodii bicarb.	gr. v	.3 Gm.
Pulv. ipecac.	gr. ss	.03 Gm.
Hydrargyri chloridi mit.	gr. iij-v	.2-.3 Gm.

M. S.—To be taken at one dose and followed in about two hours by a saline cathartic, or by sodium phosphate, ʒi (4 Gm.).

After free purgation has been brought about the following should be administered:

℞. Acid. nitro-hydrochloric. dil.	♏x	.6 Cc.
Elix. taraxaci comp.	fʒ ij	8. Cc.

M. S.—To be taken about a half-hour before meals.

Malarial cases should receive appropriate doses of quinin and patients with chronic heart and lung disease should be treated according to the necessities of the individual case. In all chronic cases, rest, liquid diet, free purgation with salines or cholagogues, and cupping will be of benefit. Strychnin sulphate and sodium arseniate may also be employed internally. The hydropathic belt, so-called, may afford great comfort and considerable support. It consists of a stout muslin binder shaped to the abdomen and provided on the inner side with cross-pieces of tape which keep next to the skin, a fold of cloth wrung out of cold water, covered by a piece of waterproof cloth or oiled silk to prevent evaporation.

In acute attacks, hot applications and sinapisms may be applied over the region of the liver.

ABSCESS OF THE LIVER.

Synonyms.—Parenchymatous hepatitis; acute hepatitis; suppurative hepatitis.

Definition.—A diffused or circumscribed inflammation of the hepatic cells, resulting in suppuration, the abscesses being sometimes single, at times double; characterized by irregular febrile attacks, hepatic tenderness, and symptoms of deranged gastro-intestinal and hepatic functions.

Causes.—The exciting causes are pathogenic bacteria, particularly the ameba coli, colon bacillus, staphylococcus, and streptococcus. In most cases the portal circulation is the transmitting medium of the infection. In amebic dysentery, the microörganisms reach the liver and produce suppuration through this system of vessels. Infectious thrombi and emboli from any area drained by the portal system, when carried to the liver give rise to purulent inflammation and abscesses. This is sometimes seen in gastric and duodenal ulcers, purulent appendicitis, and similar affections of the digestive tract. Infectious emboli from ulcerative endocarditis, pyemia, pulmonic conditions, osteomyelitis, injuries, etc., may reach the liver through the hepatic artery and abscesses result. In the new-born infant, umbilical phlebitis may terminate in hepatic suppuration. Suppuration of an hydatid cyst, suppuration following impacted gall-stones, and traumatism may also be causes of liver abscesses. Dysentery is the most common of all the etiologic factors.

Pathologic Anatomy.—If the condition is the result of dysentery or injury there will be, as a rule, but one abscess and it will in most cases occupy the right lobe. In those cases due to pyemia and similar conditions, there will be multiple abscesses. As the abscess progresses it tends to rupture and may burst into the peritoneum, intestines, stomach, gall-bladder, hepatic duct or vein, pleura, or lungs or it may perforate the abdominal wall and discharge externally. After the pus has been evacuated cicatrization occurs. Sometimes the pus is absorbed and the abscess replaced by a scar, but more frequently absorption of the pus is attended by septicemia.

Symptoms.—The constitutional manifestations include irregular intermittent fever or remittent fever, chills, sweats, obstinate vomiting, gastrointestinal disorders, constipation, light-colored stools, slight jaundice, irritability of the nervous system, melancholia, anemia, leukocytosis, and in marked cases typhoid symptoms.

The local symptoms consist of hepatic enlargement upward, circumscribed bulging, pain extending to the right shoulder, tenderness, and fluctuation. When the abscess tends to burst externally, the area over it becomes hot, red, tender, swollen, and edematous.

Diagnosis.—In doubtful cases the aspirator may be employed. *Cancer* is distinguished by its longer course, history, nodular enlargement of the liver, emaciation, cachexia, and the absence of septic phenomena.

Intermittent fever is characterized by definite paroxysms, enlargement of the spleen, and the presence of malarial organisms in the blood.

Hepatic intermittent fever differs from hepatic abscess in its history of several attacks, its less serious course, biliary colic, and obstinate jaundice.

Pleural effusion on the right side may be differentiated from hepatic abscess by diminished fremitus and vocal resonance, and bronchial breathing if the lung is compressed.

Hydatid cyst differs from abscess in its slower course, the absence of septic symptoms, and the withdrawal of clear fluid and hooklets on aspiration. In the presence of suppuration, the finding of the hooklets is alone diagnostic.

Prognosis.—In traumatic and amebic abscesses when the pus can be evacuated early, a favorable termination may occur, but in pyemic and other forms the affection is fatal.

Treatment.—Palliative measures such as the administration of nutritious food, iron, quinin, strychnin, and alcohol should be prescribed. When the abscess is single and can be definitely located it should be evacuated and drained.

ACUTE YELLOW ATROPHY.

Synonyms.—General parenchymatous hepatitis; malignant jaundice; hemorrhagic icterus.

Definition.—An acute, diffused, or general inflammation of the hepatic cells, resulting in their complete disintegration; characterized by diminution in the size of the liver, deep jaundice, hemorrhages, and profound disturbance of the nervous system, terminating in death, usually, within one week.

Causes.—The affection is apparently due to the presence of some very toxic agent in the blood. It is very rare and occurs with greatest frequency in young pregnant women from the third to the sixth month of gestation. Among the other causes may be mentioned infectious diseases, alcoholic and venereal excesses, syphilis, mental excitement, and poisoning by phosphorous, arsenic, or antimony.

Pathologic Anatomy.—In the early stage there is hyperemia of the hepatic cells with a grayish exudation between the lobules. The organ becomes soft and friable and of a dull yellow color. The cells rapidly disappear being replaced by fat globules and the liver is reduced in size and weight. Hemorrhagic extravasations are present. The peritoneal covering of the liver is loose and thrown into folds. The spleen, kidneys, heart, and muscles undergo parenchymatous degeneration. The urine is loaded with bile pigment and the blood contains a large amount of urea and considerable leucin.

Symptoms.—The early stage of this condition resembles an attack of acute catarrhal jaundice, being attended at first by gastrointestinal catarrh, coated tongue, nausea, tenderness over the epigastrium, headache, quickened pulse, slight fever, and slight jaundice. Soon the jaundice deepens; the pulse becomes slow; the headache increases; and there is persistent insomnia. Within a very short period appear delirium, fever, rapid pulse, abdominal pain, "coffee grounds" vomit, tarry stools, hemorrhages trom the mucous membranes and into the skin, convulsions, drowsiness, coma, and death. The affection seldom lasts more than a week but in some cases is prolonged to two or three weeks.

The liver diminishes rapidly in size as may be shown by palpation and percussion and there is pitting on pressure in the epigastric region. The spleen is enlarged. The urine is scanty, of high specific gravity, and contains albumin, bile, bile-stained fatty casts, renal epithelium, leucin spheres, tyrosin needles, and aromatic oxyacids. Urea is diminished and may be absent.

Prognosis.—The disease always terminates fatally. Apparent recoveries nearly always imply erroneous diagnosis.

Treatment.—The treatment consists entirely in combatting the symptoms as they arise.

SCLEROSIS OF THE LIVER.

Synonyms.—Interstitial hepatitis; cirrhosis of the liver; hob-nailed liver; gin-drinkers' liver.

Definition.—An inflammation of the intervening connective-tissue of the liver, chronic in its progress, resulting in an induration or hardening of the organ, and an atrophy of the secreting cells; characterized by gastrointestinal catarrh, emaciation, slight jaundice and ascites.

Causes.—The prolonged use of alcoholic stimulants, gin, whiskey, beer, or porter is perhaps the most common cause. It may also be due to syphilis, passive congestion, and irritation of the gall-ducts. It usually occurs in men past thirty-five years of age. The uric acid diathesis may be a causal factor.

Pathologic Anatomy.—Two varieties of the affection are recognized, *atrophic cirrhosis* and *hypertrophic cirrhosis*.

Atrophic cirrhosis begins with hyperemia of the connective-tissue (Glisson's capsule) and enlargement of the liver results from the development of brownish-red connective-tissue elements. The hypertrophied connective-tissue presses upon the hepatic cells causing them to undergo fatty degeneration. As this process advances the organ becomes reduced in size and more dense, and its surface is covered with numerous small nodules ("hob-nails"). The hepatic and portal circulation is obstructed from obliteration of their respective radicles. The changes in the hepatic structure interfere with the venous circulation of all abdominal viscera, resulting in venous congestion of the stomach, pancreas, intestines, and peritoneum, and enlargement of the abdominal veins. The hepatic peritoneum is thickened and opaque, and adhesions are formed between the liver and diaphragm, gall-bladder and stomach. On section of the liver, firm fibrous tissue is found in abundance distributed between the lobules.

Hypertrophic cirrhosis occurs in younger individuals than the preceding and does not seem to be dependent upon alcohol for its production. The organ is yellowish in color and remains enormously enlarged throughout its entire course. The newly-formed connective-tissue shows very little tendency towards contraction or towards compression of any of the branches of the portal vein. The connective-tissue is developed to a greater extent within the lobules and thus produces obstruction of the biliary channels and consequent jaundice. By some observers, it is claimed that there is a new formation of biliary capillaries and a proliferation of the liver-cells.

Symptoms.—All the manifestations of hepatic cirrhosis are due to the obstruction to the portal circulation which it induces. Persistent gastrointestinal catarrh attended by anorexia, fetor of the breath, nausea, epigastric distention and distress, flatulence, and constipation are present. When accompanied by attacks of jaundice in a drinking-man, the early stage of cirrhosis of the liver should be suspected. As the condition progresses and the obstruction becomes more marked, hemorrhages from the nose, esophagus, stomach, or intestine, hemorrhoids, dilatation of the superficial abdominal veins forming the "*caput medusae*", ascites, enlargement of the spleen, and swelling and edema of the feet occur. The condition is afebrile and emaciation, localized abdominal pain, and sometimes jaundice are present.

In *atrophic cirrhosis* the liver dullness is enlarged at first, but later becomes markedly lessened. Splenic dullness is enlarged. The skin has a muddy appearance and there is gradual emaciation. The symptoms continue from two to four years, terminating fatally in about one year after the dropsy makes its appearance. The urine is scanty, high-colored, of increased density, and is loaded with urates. The quantity of urea is diminished and blood and bile-pigment may be present. The ending of the affection is marked by drowsiness, delirium, convulsions, and coma, death resulting from toxemia, exhaustion, hemorrhage, or similar conditions.

In *hypertrophic cirrhosis*, jaundice is an early and persistent symptom. Congestion of the digestive tract, enlargement of the spleen, hemorrhoids, and ascites are absent or present only in mild degrees. The liver is permanently enlarged, smooth, tender, and the seat of paroxysms of pain. The urine is bile-stained and the percentage of urea is normal. Blood is absent. The feces may be devoid of bile-pigment or may be normal. The red blood cells are diminished about one half, and there is a relative increase in the leukocytes. This form of the affection is more rapid than the preceding, terminating with acute toxemic symptoms in death, usually in one or two years.

Diagnosis.—The characteristics of hepatic cirrhosis are the history, area of liver dullness, symptoms of portal obstruction, jaundice, and the course and termination. The distinction between the two varieties depends on the area of liver dullness, and the presence or absence of portal obstruction and jaundice.

Atrophy of the liver, or the nutmeg liver, is almost always confounded with sclerosis; the former occurs most commonly with obstructive diseases of the heart and lungs, and the surface of the organ is not nodulated, nor is there a history of alcoholism.

Cancer and tubercle of the peritoneum have many symptoms akin to sclerosis. The points of differentiation are, great tenderness over abdomen, rapidly-developed ascites, rapid decline in strength and flesh, absence of jaundice, absence of long-continued dyspepsia, absence of hepatic changes on percussion, and the presence of tubercule or cancer deposits in other organs.

Prognosis.—The outlook is unfavorable and while many cases have a very long course, the disease ultimately has a fatal termination.

Treatment.—The diet should be restricted to milk and similar unirritating food. Fatty and saccharine substances should be eliminated. Alcoholic individuals should be advised to cease drinking. The gastrointestinal catarrh should receive symptomatic treatment. Bichlorid of mercury, gr. 1-60 to 1-32 (0.002 Gm.), calomel, gr. 1-100 (0.00065 Gm.), gold and sodium chlorid, gr. 1-20 (0.003 Gm.), sodium phosphate, ʒss-i (2-4 Gm.), and potassium iodid, gr. x (0.6 Gm.), three times daily, are highly recommended. The portal congestion is best relieved by salines, such as Hunyadi, Saratoga, Friedrichshalle, or Carlsbad waters, and Rochelle or Epsom salts. The abdominal dropsy or ascites will require the administration of saline purgatives and diuretics. A half ounce of a concentrated solution of magnesium sulphate taken daily before breakfast is of value, as is also the pill containing one grain each of calomel, digitalis, and squill when given after each meal. Acetate of potassium may also be used. Tapping is necessary when these measures fail.

Surgical treatment, consisting of suturing the omentum to the margin of the abdominal incision with a view to establishing an anastomosis between the portal and systemic circulation, has been employed, but should never be considered in the presence of complicating renal or cardiac disease.

AMYLOID LIVER.

Synonyms.—Waxy liver; lardaceous liver; scrofulous liver; albuminous liver.

Definition.—A peculiar infiltration into, or a degeneration of, the structure of the liver, from the deposit of an albuminoid material which has been termed amyloid, from its superficial resemblance to starch granules.

Causes.—The principal cause is prolonged suppuration, especially of bones. It is seen in coxalgia, pulmonary tuberculosis, syphilis, rachitis, cancer, leukemia, and certain infectious diseases.

Pathologic Anatomy.—The liver is uniformly enlarged and its surface presents a pale, glistening appearance. It has a doughy consistency, and its edges are blunt. The surface of a cut section is whitish, anemic, and homogeneous. The deposit begins in the arterioles and capillaries and spreads to the fibrous tissue and parenchyma. The other viscera becomes ultimately affected by the degenerative change.

The reaction with iodin and sulphuric acid affords a certain test for the amyloid or albuminoid deposits. After further cleansing, brush over the parts a solution of iodin with iodid of potassium in water, when they will assume a mahogany color, and if diluted sulphuric acid be added, a violet or bluish tint is produced.

It may also be detected by adding a one percent solution of anilin violet, which strikes a pink color with the amyloid material, while the unaffected tissues are stained blue.

Symptoms. — There are no characteristic manifestations. Hepatic dullness is increased and there is prominence of the hepatic area. Pain is absent. The spleen and kidneys are enlarged and the urine is increased in amount, pale, albuminous, and contains amyloid casts when the kidneys are involved by the amyloid change. Disorders of digestion, diarrhea, emaciation, and anemia are common. Jaundice and ascites are infrequent.

Diagnosis.—*Leukemia* is also characterized by uniform enlargement of the liver and spleen, but the history, the examination of the blood, and the presence or absence of amyloid casts in the urine will aid in making the diagnosis.

Prognosis.—The progress of the affection may be retarded and the symptoms relieved if the underlying cause can be removed, otherwise the prognosis is unfavorable.

Treatment.—The focus of suppuration, which induces the condition in most cases, should receive prompt surgical treatment. Tonics, such as iron, syrup of the lactophosphate of lime, cod-liver oil, quinin, etc., should be administered over a long period. Da Costa recommends ammonium chlorid, gr. x-xx (0.6-1.3 Gm.) three times daily for several weeks, after which it is replaced by the syrup of the iodid of iron, beginning with ℳx (0.6 Cc.) and increasing it to ʒi (4 Cc.), for a similar period, when the first named drug is again employed.

HYDATID CYST OF THE LIVER.

Synonym.—Echinococcus of the liver.

Definition.—A cystic condition of the liver, due to the invasion and subsequent development of the embryos of the *Tænia echinococcus,* an intestinal parasite found in dogs, wolves, and jackals in Iceland, Australia, and portions of Europe. It is rare in this country, but in regions where the relation of men and dogs is more intimate, it is rather common. The ova are accidentally ingested with the food and drink by men, and on being liberated in the stomach and intestines the larvæ find their way into the portal circulation, and thus reach the liver. Here they become lodged and loose their hooklets, developing into a cyst. The cyst-wall has two layers, the inner of which is the germinal layer from which daughter-cysts are developed. The attendant irritation gives rise to the formation of an additional capsule of connective-tissue. The contents of the cyst include a clear non-albuminous fluid of low specific gravity rich in chlorids, larvæ, hooklets, and daughter-cysts. The cyst grows slowly, and on the death of the parasite it may undergo inspissation and calcification or suppuration.

Symptoms.—Unless the cyst is large there are no symptoms as a rule. The liver is then irregularly enlarged and there is a sense of fullness in the hepatic region. Fluctuation may be detected in some cases. If the cyst is near the surface, the placing of one hand over the tumor and tapping it lightly with the fingers of the other hand, will elicit a vibrating or trembling movement (hydatid thrill or fremitus). Aspiration should always be performed, as the presence of a few hooklets in the clear fluid withdrawn is diagnostic. Jaundice, pain, dyspnea, fever, and pyemic symptoms may occasionally be present. Suppuration and rupture are the most common terminations, but the possibility of such a condition remaining quiescent, should be remembered.

Diagnosis.—The history, slow course, smooth elastic fluctuating tumor, without fever or emaciation, and the aspirated fluid and hooklets will distinguish it from abscess, cancer, or other conditions with which it may be confused.

Prognosis.—In the absence of complications, the outlook is gaurdedly favorable, otherwise it is extremely serious.

Treatment.—There is no medicinal treatment. Aspiration may be performed, or the cyst may be treated as an abscess.

SYPHILIS OF THE LIVER.

Syphilis of the liver may be congenital or acquired. The congenital form may be either a diffused cellular infiltration which produces at first enlargement and hardening, and later atrophic changes and irregularities, or a circumscribed lesion, a gumma. The acquired variety includes diffuse interstitial hepatitis, gumma, amyloid disease, and endarteritis.

Symptoms.—Jaundice in the course of syphilis should always indicate subsequent careful attention to the liver. Frequently the lesions escape detection ante-mortem. The symptoms, when present, are those of portal obstruction as in ordinary cirrhosis.

Diagnosis.—This is extremely difficult and depends largely on the history and the results of the therapeutic test.

Treatment.—Antisyphilitic treatment should be promptly instituted if there is the slightest possibility of syphilis, as in the early cases the best results are obtained.

HEPATIC CANCER.

Synonym.—Carcinoma of the liver.

Definition.—A peculiar morbid growth, progressively destroying the hepatic tissue; characterized by disorders of digestion, anemia, emaciation, jaundice, and ascites, and terminating in death of the patient.

Causes.—It may arise as a primary growth, or it may be secondary to a similar affection in some adjacent or remote portion of the body. It usually occurs in men from forty to sixty years of age, and seems to be influenced by heredity, traumatism, and various forms of irritation.

Pathologic Anatomy.—In most cases the growth is secondary, and is an admixture of medullary and scirrhus cancer. It arises from the lodgement of cancerous emboli in the portal capillaries. These emboli proliferate, and cause portal obstruction and infiltration of the liver with numerous grayish-white nodules, which, when superficial, are umbilicated. The liver is increased in size. The hepatic cells atrophy, and the branches of the hepatic artery enlarge and permeate the growth. The peritoneum is adherent, cloudy, and thickened. Primary cancer of the liver occurs usually as a solitary growth, but may be nodular and accompanied by cirrhosis.

Symptoms.—The development of hepatic cancer is preceded by a history of dyspepsia, flatulency, and constipation. Abdominal distress, weight, and pain, increased on pressure, are noticed. In addition there are jaundice, ascites, occasionally intense hemorrhages, emaciation, feebleness, anemia, cold, dry, harsh skin, pinched features, dejected expression, and all the symptoms of cachexia. Fever is absent, except when there are complications, and towards the termination of the disease. The hepatic dullness is increased, and the liver is indurated, irregular, nodulated, and painful on palpation.

Diagnosis.—The age, sex, history of primary growth, usually in stomach, cachexia, pain and tenderness on palpation, and enlargement and nodulation of the liver are the distinguishing features of hepatic cancer.

Prognosis.—The disease always terminates in death, usually within a year after its recognition.

Treatment.—The treatment is entirely symptomatic. Opium will be frequently required to relieve the pain.

SARCOMA OF THE LIVER.

Sarcoma of the liver is nearly always secondary, arising, usually, as metastatic growth from melanotic sarcoma of the eye. It is multiple in most cases, and is said never to be attended by ascites. It gives rise to irregular enlargement of the liver and a host of symptoms common to all chronic hepatic affections. The diagnosis is difficult and the condition terminates fatally.

DISEASES OF THE KIDNEYS.

THE URINE.

The *normal quantity* of urine voided, varies from forty to fifty ounces (1200-1500 Cc.) in the twenty-four hours; it is decreased by free perspiration and increased by chilling of the skin.

Within the twenty-four hours, the least urine is passed during the night or in the early morning, the greater portion being passed during the course of the day.

An increase in the quantity of urine excreted, is termed *polyuria,* and may be transient or permanent. Transient polyuria follows the crisis of febrile affections, chilling of the skin, the administration of diuretics, the ingestion of large quantities of fluids, and similar conditions, while permanent polyuria results from diabetes mellitus, diabetes insipidus, chronic interstitial nephritis, and amyloid disease.

A diminution in the urinary secretion may be due to draining of the fluids of the body through other channels, as in perspiration and diarrhea, congestion, and inflammation of the kidneys, fever, collapse, hysteria, or mechanical obstruction somewhere in the genitourinary tract. When complete it is termed suppression of the urine or *anuria.*

The *normal color* is light amber, due to urobilin; the color deepens if the quantity voided be decreased, and *vice versâ.* In nearly all normal urine a cloud of mucus forms after standing a short time.

The *normal reaction* is slightly acid, due to the acid sodic phosphate, uric and hippuric acids. After meals, it may be neutral or even alkaline.

The *normal specific gravity* varies from 1.015 to 1.02; it is low when an increased quantity is passed, and high when the quantity is diminished.

The *normal odor* of urine is a peculiar, well-known, aromatic one; it is altered by certain foods, for instance, the violet stench after eating asparagus, and the garlicky odor after using garlic.

The most important organic and inorganic solid constituents held in solution, are *urea* (the index of nitrogenous excretion), from 308 to 617 gr. daily; *uric acid,* from 6 to 12 gr.; *urates of sodium, ammonium, potassium, calcium,* and *magnesium,* from 9 to 14 gr.; *phosphates of sodium, etc.,* from 12 to 45 gr., and *chlorid of sodium,* etc., from 154 to 237 gr. daily.

Urea may be increased in febrile affections, diabetes, and acute inflammatory conditions, and after the ingestion of excessive quantities of albuminous food and certain drugs. It may be determined qualitatively by the addition of nitric acid to urine which has been evaporated to about one-sixth its original volume. Crystals of urea nitrate will then be formed. For the quantitative determination of urea in the urine, two tests are employed, Davy's and Fowler's tests.

Davy's Hypobromite of Sodium Test.—Fill a graduated glass tube one-third full of mercury, and add one-half dram of the 24 hours' urine; then fill the tube evenly full with a saturated solution of hypobromite of sodium, and close it immediately with the thumb; invert the tube and place its open end beneath a saturated solution of chlorid of sodium; the mercury flows out and is replaced by the solution of salt; nitrogen gas is disengaged from the urea in the upper part of the tube. Each cubic inch of gas represents 0.645 gr. of urea in the half-dram, from which the amount passed in 24 hours may be calculated.

Fowler's Sodium Hypochlorite Test.—This test depends upon the reduction in density, caused by the decomposition of urea in solu-

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tion by sodium hypochlorite. In a mixture of one volume of urine and seven volumes of sodium hypochlorite solution, a loss of one degree in specific gravity represents the decomposition of 0.77 percent of urea. The specific gravity of the urine should always be taken first. The specific gravity of the mixture of the urine and the hypochlorite solution (Labarraque's solution) should then be ascertained by multiplying that of the pure sodium hypochlorite solution by seven, adding to this the specific gravity of the urine, and dividing by eight. The mixture (one part urine and seven parts hypochlorite solution) is set aside for about two hours to allow complete decomposition, when the specific gravity is again taken and compared with that of the urine.

A diminution in the quantity of urea usually indicates deficient elimination, and is observed in nephritis, especially in the late stages, cachectic conditions, acute yellow atrophy of the liver, hepatic cirrhosis, diarrhea, acute gout, chronic rheumatism, leprosy, pemphigus, melancholia, catalepsy, hysteria, and after free perspiration, fasting, and a vegetable or milk diet.

Uric acid or urates in the urine constitute the condition known as *lithuria*. When in excess in the urine they are precipitated by cold as brickdust deposits. Their quantity is increased in indigestion, gout, fever, wasting diseases, malaria, scurvy, diabetes, rachitis, and after free perspiration and diarrhea, and the ingestion of nitrogenous foods, colchicum, salicylic acid, and corrosive sublimate. They are diminished in amount in an acute attack of gout, anemia, chlorosis, chronic nephritis, and after the use of drugs, such as caffein, lithia, potassium iodid, etc.

Tests for Uric Acid and Urates.—The urine should be evaporated to dryness on a water-bath and covered with strong nitric acid, after which the mixture is again evaporated. When cool a drop or two of ammonium hydroxid is added to the residue, whereupon a beautiful red color will be produced by the formation of murexid or ammonium purpurate.

The contact test consists in pouring nitric acid slowly down the side of a test-tube containing a small quantity of urine. At the junction of the two liquids, a yellowish red zone will be formed by the uric acid, while above this will be a dense milky zone of acid urates, which dissolves on agitation.

For the quantitative determination of uric acid, three ounces of the 24 hours' urine (after being slightly acidulated, boiled, and filtered while hot) should be mixed with one-tenth as much nitric acid and placed in a cool place for twenty-four hours. The uric acid crystals are then collected on a weighed filter, washed, and dried at 212° F. The increased weight represents the quantity of uric acid in three ounces of urine.

Phosphates occur in the urine as ammoniomagnesium or triple phosphates, and as crystalline and amorphous phosphate of lime. They are precipitated in alkaline urine and often produce a cloudiness when the urine is heated, which disappears on the addition of nitric or acetic acid. The addition of an alkali, such as ammonium hydroxid, to urine containing amorphous phosphates causes their precipitation. Triple phosphate may be recognized under the microscope by its large rhombic or "coffin-lid" shaped prisms, which are freely soluble in acetic acid. Crystalline phosphate of lime is a rare form and appears as rods or needles, sometimes grouped together as sheaves or stars, which are also soluble in acetic acid. An excess of phosphates in the urine constitutes *phosphaturia,* and occurs in rachitis, osteomalacia, gout, nervous dyspepsia, and various nervous affections. They are apparently

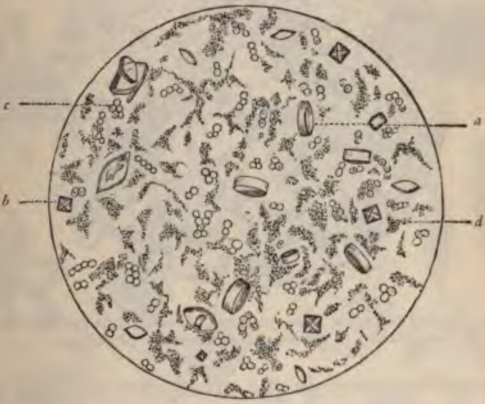

FIG. 7.— SOME DEPOSITS IN ACID FERMENTATION OF THE URINE.—(*Landois.*) *a.* Bacteria. *b.* Amorphous sodic urate. *c.* Uric acid. *d.* Calcium oxalate.

in excess in alkaline urine. Triple phosphate in combination with amorphous phosphates, bladder epithelium, and pus cells in freshly voided urine indicates cystitis.

The *magnesium test for phosphates* consists in the addition of a mixture of one part each of magnesium sulphate, ammonium chlorid, and liquor of ammonia and eight parts of distilled water to three times as much urine, whereupon a cloudy, milky precipitate will be formed which will be creamy if the phosphates are in excess.

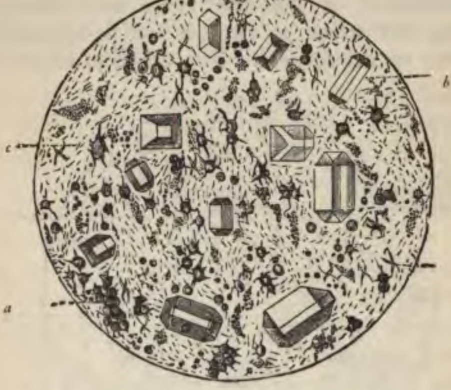

FIG. 8.— SOME DEPOSITS FROM AMMONIACAL URINE (ALKALINE FERMENTATION). *a*. Acid ammonium urate. *b*. Ammoniomagnesium phosphate. *c*. Basteria (Landois).

Chlorids are increased in the urine after exertion of any kind and during absorption, and are diminished in pneumonia, febrile affections, and kidney and wasting diseases. To test for their presence, albumin should first be removed by nitric acid, or boiling and filtration, after which one drop of silver nitrate solution (one part to eight) should be added. The presence of chlorids will be indicated by a white precipitate of silver chlorid.

Oxalates are recognized only by microscopic examination, and occur as dumb-bell-shaped crystals or octahedral crystals. Their presence is termed *oxaluria*, and indicates impeded metamorphosis. It is encountered in diabetes, after the ingestion of pears, rhubarb, spinach, and similar substances, in certain forms of indigestion, in gout, and in certain nervous affections. It is accompanied by pains in the back and loins, flatulence, dyspepsia, hypochondriasis, and melancholia.

Cystin is a rare sediment sometimes observed in the urine of children and young male adults, and in occasional instances it forms a basis for a calculus. It occurs as hexagonal prisms, which may be superimposed upon each other or grouped in irregular masses.

Leucin appears in the urine as highly refracting spheres, which have a radiating arrangement and are insoluble in ether. They are usually combined with **tyrosin** crystals, which are long and needle-like, in acute yellow atrophy of the liver and phosphorous poisoning. Tyrosin may also be found in typhoid fever.

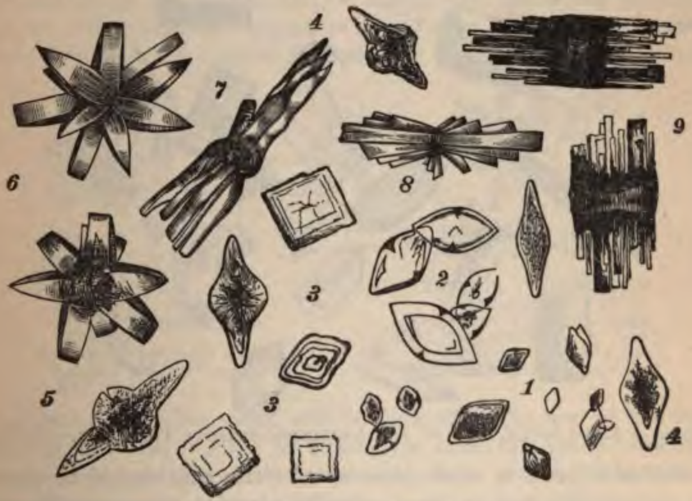

Fig 9.— Some Forms of Uric Acid.
1. Rhombic crystals. 2. Whetstone forms. 3, 3. Quadrate forms. 4, 4, 5. Irregular forms. 6, 8. Groupings into roset forms. 7, 9. Bundle forms, precipitated by adding hydrochloric acid to the urine. The crystals may be pigmented or lightly colored brownish-yellow; the pigment is presumed to be uroerythrin, also called urochrome.— (*Landois.*)

Cholesterin plates may be encountered in the urine in jaundice, chyluria, fatty degeneration of the kidneys, and diabetes.

Mucus alone is not visible, but induces cloudiness from having entangled and precipitated mucous or pus corpuscles, epithelium, and various crystals. To detect its presence a few drops of acetic acid, alone or combined with the compound liquor of iodin are added to the urine, thereby rendering visible threads and bands of mucin which are dissolved on the addition of nitric acid.

Albumin occurs in the urine usually in the form of serum albumin, but other proteids may also be found. It is encountered in congestion and inflammation of the kidneys, anemic conditions, pregnancy, acute febrile diseases, diarrhea, cholera, certain nervous diseases as meningitis, cerebral hemorrhages, epilepsy, etc., and in healthy adults after exertion, exposure, or a rich diet. Contamination of the urine with blood or pus, anywhere along the genito-urinary tract produces the form known as extrarenal albuminuria.

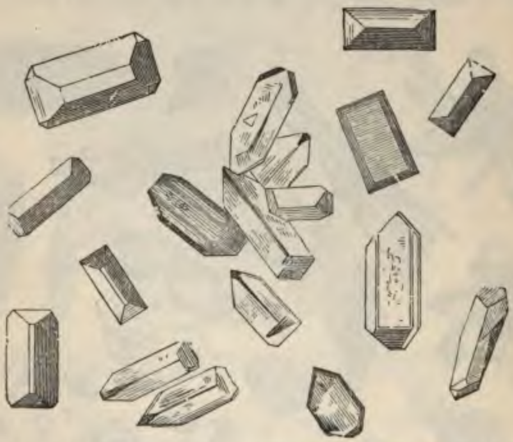

FIG 10.— FORMS OF CRYSTALS OF THE AMMONIOMAGNESIUM PHOSPHATE.—(*Tyson.*)

Heller's Test.—A small quantity of nitric acid should be placed in the test-tube and an equal quantity of urine superimposed upon it by means of a pipette. A white zone at the line of junction will result if albumin is present. A diffuse pink ring, slightly above the line of contact, indicates the presence of uric acid. Balsam of copaiba, oleoresin of cubebs, turpentine, and similar drugs, when ingested, give rise to the same reaction in the urine as albumin, but their rings are dissolved by the addition of alcohol.

Heat and Nitric Acid Test.—The urine is slightly acidulated and boiled. A white deposit, which is not dissolved by the addition of nitric acid, drop by drop, is due to coagulated albumin. An excess of the acid will cause solution of the precipitate.

Johnson's Picric Acid Test.—Filtered urine should be placed in a test-tube and a saturated solution of picric acid is added, drop by drop; in the presence of albumin an opaque white cloud will be formed, which is rendered more marked on the application of heat.

Robert's Nitric-Magnesium Test.—The reagent is prepared by mixing one part of pure nitric acid with five parts of a saturated solution of magnesium sulphate. The test is made in the same manner as Heller's test; a compact, dense, white layer at the line of contact indicates the presence of albumin.

Potassium Ferrocyanid Test.—A solution of potassium ferrocyanid (1 to 20) should first be mixed with about one-third its bulk of acetic acid and placed in a test-tube. The urine is then gently laid over the reagent by means of a pipette, and if albumin is present a milk-like cloud will be formed at the line of contact. The test may also be made by mixing the urine and the ferrocyanid solution first, and then adding the acetic acid, drop by drop, when the milkiness will appear throughout the mixture if the test is positive. The urine may also be thoroughly admixed with the reagent in the former method, resulting in a similar diffuse cloudiness.

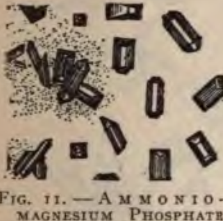

FIG. 11.—AMMONIO-MAGNESIUM PHOSPHATE (TRIPLE PHOSPHATE).—(*Gould.*)
Triangular prisms with beveled edges; "coffin-lid" crystals.

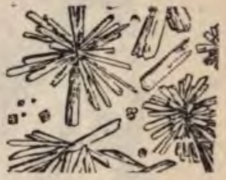

FIG. 12.—AMORPHOUS GRANULES, WEDGE-SHAPED CRYSTALS, SOME ARRANGED IN ROSETS OF CALCIUM PHOSPHATE.—(*Gould.*)

Tanret's Potassium-Mercuric Iodid Test.—This is a contact test similar to Heller's test. The reagent employed is made up of potassium iodid, 3.32 Gm., bichlorid of mercury, 1.35 Gm., Acetic acid, 20 Cc., and distilled water, 30 Cc. The potassium iodid and the bichlorid of mercury should be dissolved separately and their solutions mixed, after which acetic acid is added and the entire mixture made up to 60 Cc. with distilled water.

Millard's Test.—This is an extremely delicate contact test and requires considerable care in its employment. The fluid consists of carbolic acid, 8 Cc., glacial acetic acid, 28 Cc., and the solution of potassium hydrate, 84 Cc.

Quantitative Test.—For the determination of the quantity of albumin, Esbach's albuminometer is most convenient. The tube should first be filled up to the mark "U" with urine. The reagent, consisting of picric acid, 10 Gm., citric acid, 20 Gm., and water, one liter, should be poured over the urine until the mark "R" is reached. The rubber stopper is then inserted and the contents of the tube thoroughly admixed by shaking. The tube is then set aside for 24 hours, when a precipitate will have formed and its quantity will indicate on the graduated scale on the tube, the number of grams of albumin to the liter.

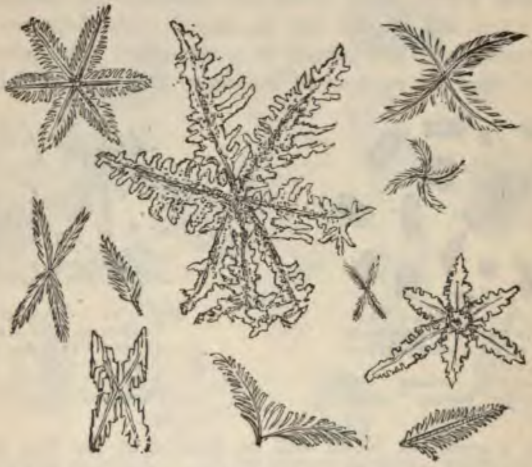

FIG 13. — STELLATE AND FEATHERY CRYSTALS OF TRIPLE PHOSPHATE.—(*Tyson.*)

Sugar in the urine or *glycosuria,* may be present normally, after the ingestion of large quantities of saccharine substances, but is usually present as an abnormal constituent in diabetes mellitus, and diseases of the pancreas. It may also occur in diseases or injuries of the floor of the fourth ventricle, certain nervous diseases, pregnancy, and poisoning by drugs, such as chloroform, nitrites, etc.

Moore's test for sugar consists in boiling the urine with half its volume of sodium or potassium hydroxid solution (10 percent). Should a white flaky precipitate of earthy phosphates be formed, it should be removed by filtration and the urine again boiled. If glucose is present, the liquid then becomes brown and finally black, due to the formation of glucic and finally melassic acid.

Boettcher's bismuth test requires first, the addition of the urine to half its volume of sodium or potassium hydroxid solution, after which a small portion of bismuth subnitrate is mixed with the resultant liquid and the whole shaken together and boiled. The presence of sugar reduces the salt, and black metallic bismuth is deposited. If there is but little glucose, a grey precipitate is formed. If there is any reason to suspect the presence of albumin, it should be removed before applying this test, as it interferes by forming bismuth sulphid, which is also black.

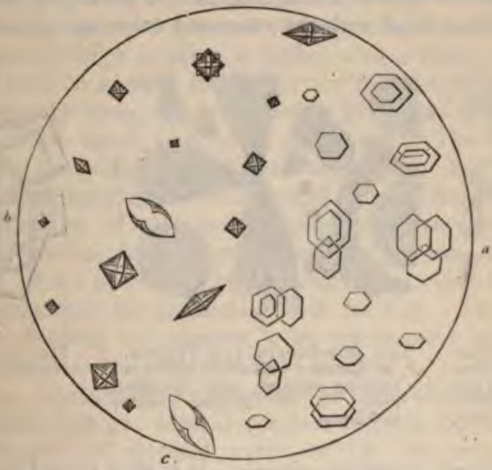

FIG. 14.

a. Crystals of cystin. *b.* Crystals of oxalate of lime. *c.* Hour-glass forms of *b.* (*Landois.*)

Johnson's picric acid test is applied by adding a few drops of a saturated solution of picric acid to urine, which has been previously rendered alkaline by means of a sodium or potassium hydroxid solution, and boiling the mixture, which then becomes claret-red in color in the presence of glucose. Creatinin gives a similar reaction.

Trommer's test is performed by adding to the urine a few drops of a solution of cupric sulphate, and then its own volume of a potassium hydroxid solution, after which the entire mixture is boiled. The immediate formation of a yellowish precipitate of hydrated cupric suboxid denotes the presence of sugar.

The *Phenyl-hydrazin test* requires for its reagent, a mixture of two grains of phenyl-hydrazin hydrochlorid and three grains of sodium acetate dissolved in half a test-tube full of water, by heating. The test-tube is then filled with the suspected urine and placed in boiling water for fifteen or twenty minutes. It is then placed in cold water. If the sugar is present in large amounts, a yellow precipitate of needle-like crystals may be observed with the unaided eye, but ordinarily the microscope is necessary to detect these crystals of phenylglucosazon.

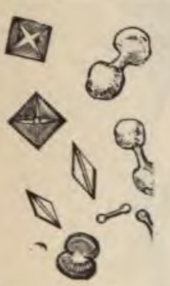

Fig. 15.—Dumb-bell and Octahedral Crystals of Calcium Oxalate.—(*Gould*.)

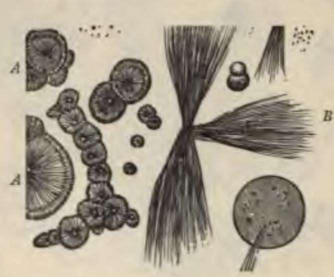

Fig. 16.—Leucin and Tyrosin. *A, A.* Leucin; yellowish, highly refracting spheres, with radiating lines. *B, B.* Tyrosin, needles and sheaf.

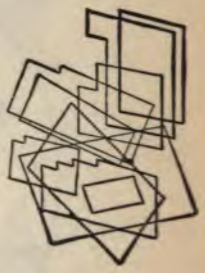

Fig. 17.—Cholesterin.—(*Landois*.)

The *fermentation test* depends upon the breaking up of glucose into alcohol and carbon dioxid, by the action of the yeast ferment. In the practical application of this test, a bottle or test-tube is filled with urine and a very small piece of compressed yeast is added, and the bottle or tube corked. The mixture is placed in a chamber at from 70° F. to 100° F. for about ten hours. In the presence of sugar, there will be a loss in specific gravity, and carbon dioxid will be evolved and collect at the top of the bottle or tube.

Quantitative Tests for Glucose.—Fehling's test necessitates fresh preparation of the reagent before its application. Two solutions are employed: the first consists of 34.652 grams of pure cupric sulphate dissolved in distilled water and diluted up to 500 cubic centimeters; the second consists of 175 grams of pure Rochelle salt and 60 grams of caustic soda, dissolved in 400 cubic centimeters of distilled water and boiled, after which it is made up to 500 cubic centimeters with distilled water. The Fehling's solution proper, is made by mixing equal quantities of the foregoing solutions, and its preparation is such that one cubic centimeter of it will reduce 0.005 gram of glucose. In applying this test, 1 cubic centimeter of Fehling's solution is boiled with 4 cubic centimeters of distilled water to test it. If the solution remains clear the urine is added, drop by drop, by means of a graduated pipette, from which two drops equal 1-10 cubic centimeter. The number of drops necessary to change all of the blue color of the solution to yellowish red, on boiling, should be noted and divided by two, thus giving the number of 1-10 cubic centimeters required. If one cubic centimeter has been required for this purpose, it indicates the presence of 0.5 percent of sugar, and if two cubic centimeters have been employed the percentage will be 0.25. This result is obtained by dividing the number 5 by the number of tenths of urine required to reduce the Fehling's solution.

Pavy's test requires the employment of the following solution: cupric sulphate, 320 gr., neutral potassic tartrate, 640 gr., caustic potash, 1280 gr., and distilled water, 20 f ℥. In performing the test 200 minims, or a multiple of this quantity, of the solution should be boiled in a porcelain dish and the urine added minim by minim. A yellowish precipitate points to the presence of sugar. The quantity of urine necessary to entirely decompose the solution should then be noted. The quantity of sugar may then be deduced by recalling the fact that one grain of glucose is necessary to reduce 200 minims of the copper solution.

Robert's differential density test consists in taking two measured specimens of the urine, to one of which is added a small piece of yeast. Both are placed in a chamber at a temperature from 75° to 80° F. for 24 hours, after which the specific gravity of each is taken. The presence of sugar will cause a loss of specific gravity, and the number of degrees lost will correspond approximately to the number of grains of sugar in each ounce of urine.

Blood.—The presence of blood in the urine may be due to congestion, inflammation, tumors, or traumatism of any portion of the genitourinary tract, blood diseases such as hemophilia, scurvy, anemia, etc., vicarious menstruation, calculi, parasites, etc. Hemorrhage from the urethra is indicated when the blood is present in the first urine passed, while the appearance of blood in the last portions points to some bladder condition. Blood from the kidneys appears as blood-casts, or clots, or freely mixed with the urine.

Tests for Blood.—Heat and nitric acid causes a deposition of the albumin of the blood, with changing of its coloring matter to a dirty brown.

Heller's test consists in boiling the urine with caustic potash, thus causing a precipitation of the phosphates and coloring matter of the blood, which deposit appears of a blood-red color when viewed by transmitted light.

The microscope and spectroscope should also be employed as confirmatory tests.

Bile.—The presence of biliary pigments may be crudely determined by agitating the urine, whereupon a yellow foam is produced. A simple test is to filter the urine through white filter paper; then place the paper on a porcelain dish or plate and add a drop of strong nitric acid. In the presence of bile pigment, concentric rings of red, violet, blue, and green, will be formed at the line of contact.

Heller's test is performed as follows: Pour into a test-tube about 1.6-fʒ of pure hydrochloric acid, and add to it, drop by drop, just sufficient urine to distinctly color it. The two are mixed. Then drop down the side of the tube pure nitric acid, which will "underlay" the mixture of hydrochloric acid and urine. At the point of contact between the mixture and the colorless nitric acid a handsome "play of colors" appears. If the "underlying" nitric acid is now stirred with a glass rod, the set of colors which were superimposed upon one another will appear alongside of each other in the entire mixture, and should be studied by transmitted light. If the hydrochloric acid, on addition of the biliary urine, is colored reddish-yellow, the coloring matter is ***bilirubin;*** if it is colored green, it is *biliverdin*.

Pettinkofer's test consists first in the addition of a few grains of cane-sugar and a drop or two of sulphuric acid to the urine, after the entire mixture is boiled. The formation if a violet-red color indicates the presence of biliary pigments.

Pus.—The presence of pus in the urine is termed *pyuria* and usually indicates suppuration along the genitourinary tract. Its source may be detected to a great extent by the time of its appearance in the flow, as with blood in the urine. When present in the early part of micturition the urethra is usually diseased, if at the end and in alkaline urine the trouble is in the bladder, but if it is freely admixed with an acid or neutral urine, the probabilities are that the kidneys are at fault. The addition of an equal quantity of a solution of potassium hydroxid to urine containing pus gives rise to the formation of a viscid gelatinous mass. The microscope may also be employed to detect pus.

Acetone.—Acetone occurs in the urine in the advanced stages of diabetes, in starvation, in cancer, in autointoxications, in digestive disturbances, in fevers, in certain psychoses, and to a very slight extent in health.

Legal's test for its detection consists in the addition of a few drops of a strong solution of sodium nitroprussid to about 4 Cc. of urine, which has been previously rendered alkaline by potassium hydroxid solution. In the presence of acetone a red color is produced, which turns purple on the addition of a few drops of acetic acid.

It may also be detected by the precipitation of iodoform, which occurs when urine containing it is mixed with a few drops of iodo-potassium iodid solution and sodium hydroxid solution.

Diacetic Acid.—Diacetic acid occurs in children in fevers, in diabetes, and in autointoxications. Coma usually follows its appearance. To detect its presence the urine should be boiled with a solution of ferric chlorid and if diacetic acid is present, a Burgundy-red color will be produced.

Indican.—The presence of indoxyl-potassium sulphate or indican, in the urine is termed *indicanuria*. It is a sign of intestinal putrefaction and is observed after the ingestion of an animal diet, in constipation, in diarrhea, and in intestinal tuberculosis. It also accompanies decomposition of albumin in cavities, and is encountered in empyema and puerperal peritonitis.

Jaffe's test consists in mixing 10 Cc. of strong hydrochloric acid with an equal volume of urine, from which albumin has been tested for and removed, and while shaking add, drop by drop, a freshly prepared saturated solution of chlorid of lime. Chloroform is then added, which dissolves out an indigo-blue substance if indican is present.

Another method sometimes employed requires the addition of 20 drops of urine to 4 Cc. of hydrochloric acid. If the proportion of indigo be slightly above normal, the resultant color will be rather light yellow; if in excess the acid will turn violet or blue—the color being more intense the greater the quantity. If no coloration appears within a minute or two, there is no excess of indican.

Peptone.—Peptone may be encountered in the urine in jaundice, hepatic cancer, acute miliary tuberculosis, scarlet fever, and typhoid fever.

Rolfe's test is performed by adding 4 Cc. of Fehling's solution to a small quantity of urine by means of a pipette. Peptone will be indicated by a rose-colored halo immediately above the line of contact.

Ehrlich's Diazo-reaction.—The presence of aromatic substances in the urine, such as occur in typhoid fever, pneumonia, measles, tuberculosis, and septic infection may be detected by the following test:

I. Take 2 Gm. (30 gr.) of sulphanilic acid, 50 Cc. of hydrochloric acid, and 1000 Cc. of distilled water.

II. Take solution sodium nitrite in water of the strength of 0.5 per cent. Place fifty parts of No. I and one part No. II in a test-tube and add equal amounts of urine. The entire mixture is rendered strongly alkaline by strong ammonia water.

If the diazo-reaction occurs the mixture becomes carmine red; now shake the tube, and if the red color is seen in the foam, the test is complete. Allow the tube to stand a day and a green precipitate forms.

Microscopic Examination.—For the determination of substances by the microscope, other than the crystals already mentioned, it is necessary to produce first, a sediment by means of the centrifuge, or in its absence, by allowing the urine to stand for 12 or 24 hours after having added ten drops of chloroform, five grains of chloral, formalin, or a few drops of carbolic acid to prevent decomposition.

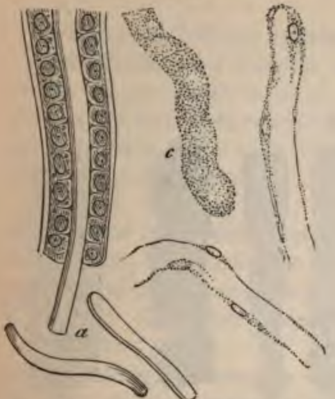

FIG. 18.—ILLUSTRATING THE FORMATION OF CASTS.—(*Ringfleisch.*) *a.* Hyaline casts in place. If it comes away bringing nothing with it, it will remain a hyaline cast. If it brings epithelium, it will be an epithelial cast; if the epithelium is granular, it will be a granular cast; if fatty, a fatty cast. *c.* Granular cast. The two casts in the lower corner, and to the left, are hyaline; the remaining casts are largely hyaline, but bear a few epithelial cells.

Tube-casts should always be carefully sought for in the sediment. They are moulds of the uriniferous tubules and vary in character according to the existing abnormal condition of the kidneys. Usually they are composed of albuminoid substances, but there may be in addition epithelium, degenerated cells, blood corpuscles, or fat globules. Their length is about 200 microns or more, and their width from 4 to 40 microns. While usually straight, they may be curved or twisted upon themselves.

Blood-casts are composed of coagulated blood and blood cells, and point to the presence of some hemorrhagic condition of the kidney. Often the cast is in reality a hyaline cast embedded with blood corpuscles.

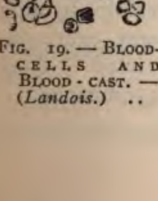

FIG. 19.—BLOOD-CELLS AND BLOOD-CAST.—(*Landois.*) ..

Epithelial casts may also be considered as hyaline casts covered and infiltrated with epithelial cells. They denote desquamation, and are seen in the urine and in acute parenchymatous nephritis.

Fatty casts are those in which the coagulated material forming the moulds of the tubules is studded with oil globules. They indicate fatty degeneration of the kidney and occur in chronic pareninterstitial nephritis.

Granular casts are made up of coagulated material and granular debris. They are usually observed in contracted kidney.

Hyaline casts or mucous casts, are transparent delicate cylinders. They may occur in health, but are always observed in congestion or inflammation of the kidney.

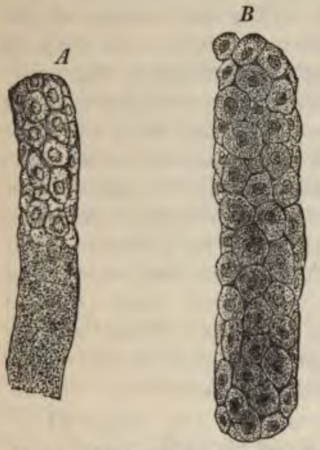

FIG. 20.—EPITHELIAL CASTS.—(*Landois.*) A. Epithelial cast, the lower end of which is coarsely granular. B. Epithelial cast in which the epithelial cells, though themselves granular, have not broken up.

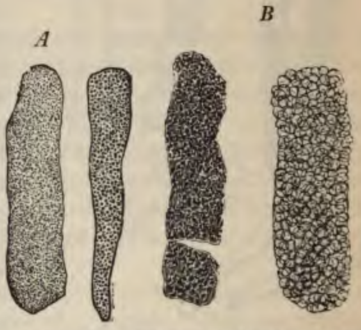

FIG. 21.—GRANULAR CASTS.—(*Landois.*)
A. Granular casts in which the granules are fine and the dissolution of the epithelial cells is complete.
B. Granular casts in which the granules are coarse and the outlines of the epithelial cells at points faintly distinguishable.

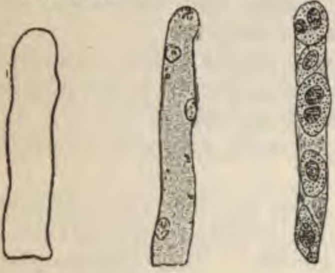

FIG. 22.

a. Hyaline cast. *b.* Hyaline cast with a few attached leokocytes. *c.* Hyaline cast with attached epithelium, truly an epithelial cast. —(*Landois*)

Pus-casts are made up of albuminous material, degenerated leukocytes, and bacteria, and are indicative of renal suppuration.

Waxy casts are large and yellowish in color, and give the amyloid reaction. They are present in chronic parenchymatous nephritis.

Cylindroids resemble hyaline casts, but are longer, more tapering and constricted. They have no clinical significance.

CONGESTION OF THE KIDNEYS.

Synonyms.—Renal hyperemia; catarrhal nephritis.

Definition.—An increase in the amount of blood in the vessels of the kidneys; when arterial, it is termed *active congestion;* when venous, *passive congestion;* characterized by pain, frequent desire for urination, and scanty, high-colored urine, occasionally containing albumin or blood.

Causes.—*Active*: cold; irritating substances eliminated by the kidneys, as turpentine, copaiba, cantharides, carbolic acid, nitrate or chlorate of potash; the eruptive or continued fevers; injuries over the kidneys; pregnancy.

Passive: obstructive diseases of the heart or lungs, pressure of the pregnant uterus.

Pathologic Anatomy.—The kindeys enlarge and increase in weight; redness increases (the color being bluish if *passive*), with points of vascularity, corresponding to the Malpighian bodies, and occasionally minute ecchymoses. The abnormal hyperemia causes a catarrhal state of the ducts of the pyramids, with shedding of their epithelium.

If mechanical (*passive*) obstruction continues for some time, increase of the connective-tissue with consequent induration and contraction results, a form of chronic Bright's disease.

Symptoms.—*Active variety*: pain over kidneys and following the course of the ureters into the testicles and penis, irritable bladder, almost constant and pressing desire for urination, the urine scanty, high-colored, and occasionally bloody, with fibrin, casts, and albumin; there is, as a rule, no pain during the act of urination. The constitutional symptoms are headache, slight nausea, vomiting, and a general feeling of discomfort. If the condition persists, inflammation of the kidney results.

Passive: the kidney changes are masked by the lung or heart trouble, until dropsy, scanty high-colored albuminous urine are observed.

Prognosis.—*Active*: if recognized and properly treated, favorable.

Passive: controlled by the cause, and if prolonged, terminating in interstitial nephritis .

Treatment.—The most important indication is to ascertain and remove the cause. Rest in bed is necessary. Liquid diet and saline purgatives should be administered. A warm bath, diaphoretics, and dry or wet cups over the loins should be employed. Infusion of digitalis and bland drinks are indicated. Irritability of the bladder may be relieved by camphor, gr. ii-iv (0.13-0.26 Gm.), every four hours, alone or combined with morphin sulphate, gr. 1-12 to 1/6 (0.005-0.011 Gm.), or by morphin hypodermically.

The treatment of the passive form resolves itself into the treatment of the cause, remembering that there is too much blood in the veins and too little in the arteries. There are three ways of restoring the circulation. By venesection, opening a large vein; by increasing the power of the heart by the use of digitalis or strophanthus, preferably the first named; and by dilatation of the capillaries with inhalations of amyl nitrite or the internal use of nitroglycerin (1 percent solution), ♏j-iij (0.06-0.18 Cc.) every four hours. The bowels should be kept soluble by salines.

ACUTE PARENCHYMATOUS NEPHRITIS.

Synonyms.—Acute Bright's disease; acute desquamative nephritis; acute tubal nephritis; acute croupous nephritis.

Definition.—An acute inflammation of the epithelium of the uriniferous tubules; characterized by fever, scanty, high-colored, or smoky urine, dropsy, with more or less constant nervous phenomena, the result of acute uremia.

Causes.—Cold and exposure, scarlatina, diphtheria, malaria, and other infectious diseases, traumatism to the back, pregnancy, and the persistent use of irritants, such as turpentine, cantharides, phosphorus, ginger, etc., are the most common causes. It may also be associated with certain skin diseases, extensive burns of the skin, and simple follicular tonsillitis. The affection is most frequent in childhood.

Pathologic Anatomy.—The kidneys are generally swollen, engorged, more vascular, and of red color; in the second stage the organ remains large, irregularly red, especially the cortex; the tubules are engorged and filled with epithelium, blood corpuscles, and fibrin. The capsule is easily detached, and is more opaque than normal. If the termination is favorable the swelling lessens, the vascularity diminishes, and the tubules gradually return to their normal condition.

Symptoms.—In mild cases, slowly developing dropsy with anemia, shortness of breath or dyspnea, and weakness are the only symptoms, the diagnosis being confirmed by the results of urinary examination. Usually, however, it begins suddenly with nausea, violent and persistent vomiting, fever, and dull pain over the kidneys, following the course of the ureters. There is a frequent desire to urinate, and diarrhea, harsh and dry skin, and a quick, tense, and full pulse are present. Dropsy soon appears, beginning first in the eyelids and face, but later becoming generalized. Anemia and weakness are marked particularly in post-scarlatinoid cases. Uremic symptoms may develop at any time during the attack. The affection lasts from one to four weeks.

The *urine* is of high specific gravity, 1025 to 1030, scanty, smoky (like beef washings) in color, due to the presence of blood. Albumin is present in large quantities, and the microscope reveals hyaline, blood, granular, and epithelial casts of the uriniferous tubules, blood corpuscles, uric acid, urates, oxalate crystals, and epithelium. The total amount of urea eliminated during the twenty-four hours is lessened from one-fourth to one-half. The amount of phosphates and chlorids is also lessened.

Complications.—Pericarditis, pleurisy, pneumonia, peritonitis, and uremia are the principal complications.

Diagnosis.—The diagnostic features of this disease are its history, the age at which it occurs, the sudden onset, the dropsy, and the urine which is scanty, smoky, and of high specific gravity, containing albumin, diminished quantity of urea, tube-casts (hyaline, blood, epithelium, and dark granular casts), blood cells, epithelium, and granular cells.

Prognosis.—The prognosis is generally favorable, recovery occurring in most cases under prompt and appropriate treatment. Uremia may, however, occur in the course of the disease and lead to a fatal termination. Pulmonary edema, purulent exudations into the serous cavities, and exhaustion, may intervene and produce death. The affection may pass into chronic nephritis.

Treatment.—The patient should be placed at rest in bed until all the symptoms have disappeared. A strictly milk diet is most suitable, but if the depression and weakness are marked, animal broths, and even oysters, may be allowed. Tea, coffee, beef-tea extracts, and stimulants should be interdicted. Water or cream of tartar lemonade may be freely used for its diuretic effect. Dry cups should be applied over the kidneys, followed by digitalis or jaborandi poultices, using equal parts of flaxseed and the leaves of digitalis or jaborandi. The bowels should be kept freely opened by means of the saline cathartics, compound jalap powder, ʒi (4 Gm.), in water before breakfast, or elaterium, gr. 1/6 (0.011 Gm.), repeated as the occasion requires. Combined with these procedures there should be free sweating or diaphoresis. This is best obtained by the use of the hot-air bath, the hot pack, or the wet sheet and blanket bath, stimulating the peripheral circulation after free sweating has occurred, by rubbing with alcohol and water. Drugs may be administered coincidently to aid the sweating process. Spirits of nitrous ether, ♏v-xxx (0.3-2 Cc.), fluid extract of pilocarpin, ♏v-xxx (0.3-2 Cc.), every three or four hours, pilocarpin hydrochlorate, gr. 1/6 (0.011 Gm.), hypodermically, as the occasion requires, or the wine of ipecac, ♏i-iii (0.06-0.2 Cc.), every half hour may be used for this purpose. Diuretics, such as digitalis, digitalin, gr. 1-100 (0.00065 Gm.), caffein citrate, gr. ii-iv (0.13-0.26 Gm.), or spartein sulphate, gr. 1/3-1/2 (0.02-0.03 Gm.) should be employed. The following formula (Millard) is suitable in the majority of cases:

℞.	Tinct. digitalis ....f℥ ss	15. Cc.
	Aceti scillæ ....f℥ iss	45. Cc.
	Spts. ætheris nitrosi....f℥ ij	60. Cc.

M. S.—Teaspoonful every two to four hours, in water.

The following combination has also given excellent results:

℞.	Potassii acetat. ....ʒ iv	15. Gm.
	Inf. digital. ....f℥ iij	90. Cc.
	Liq. potassii citratis....f℥ iij	90. Cc.

M. S.—Tablespoonful every two to four hours, in water.

Dr. James Tyson, than whom there is no greater authority in nephritic conditions, strongly urges the use of infusion of digitalis instead of the tincture. He also recommends, as an admirable diuretic combination, Trousseau's diuretic wine, viz.:

℞. Junip. contus	ʒ x	40. Gm.
Pulv. digitalis	ʒ ij	8. Gm.
Pulv. scillæ	ʒ j	4. Gm.
Vin. xerici	Oj	480. Cc.
Macerate for four days and add		
Potassii acetatis	ʒ iij	12. Gm.

Express and filter.

S.—Tablespoonful three times a day for an adult.

The onset of uremia (*q. v.*) calls for special treatment.

As soon as the blood disappears from the urine some preparation of iron, preferably Basham's mixture, should be administered until the health is entirely restored. The addition of one minim (0.065 Cc.) of spirit of nitroglycerin to each dose of Basham's mixture increases its efficiency.

℞. Liq. ammon. acetat.	f℥ vj	180. Cc.
Acid. acetic.	fʒ iij	12. Cc.
Tinct. ferri chlor.	fʒ v	20. Cc.
Alcoholis	f℥ ij	60. Cc.
Syrup	f℥ iv	120. Cc.
Aquæ	f℥ iv	120. Cc.

M. S.—Basham's mixture. Dose, one dram to one ounce, diluted.

CHRONIC PARENCHYMATOUS NEPHRITIS.

Synonyms.—Chronic Bright's disease; chronic croupous nephritis; chronic tubal nephritis; chronic albuminuria; large white kidney.

Definition.—A chronic inflammation of the cortical tubular structure of the kidneys; characterized by albuminous urine, dropsy and increasing anemia, with attacks of acute uremia.

Causes.—It rarely follows an acute attack; in the majority of cases it is subacute or chronic from the onset. It is a disease of young adults, especially males, and is seldom observed after forty years of age. Habitual exposure to cold and wet, malaria, syphilis, alcoholic excesses, chronic mercurialism, lead-poisoning, opium habit, protracted suppuration, phthisis, hepatic disorders, pregnancy, and some undetermined nervous condition are the principal causes.

Pathologic Anatomy.—The kidney is large, often twice its natural size, smooth, and white or yellowish-white in color. The capsule is nowhere adherent to the organ. Upon section, considerable tumefaction of the cortical substance and the rarity of vascular striæ are recognized. The medullary substance shows no appreciable alteration, its color being normal. The convoluted tubes are irregularly dilated and thickened, and filled with broken-down granulated epithelium and fibrinous casts. In pronounced cases there is fatty degeneration of the tubular epithelium. The intertubular matrix is greatly thickened—a change due to hyperplasia of the connective-tissue elements, to the migration of the white corpuscles and their subsequent multiplication and fatty transformation, and to a quantity of fluid exudation, the product of the increased pressure in the veins. As the affection progresses the connective-tissue tends to undergo contraction, and the organ becomes pale and reduced in size, the capsule becomes more or less adherent, and the surface of the kidney becomes uneven. During this contracting stage small hemorrhages may appear, scattered throughout the cortex.

Symptoms.—The onset of the disease is gradual and insidious, being marked by a period of ill-health, anemia, digestive disturbances, weakness, puffiness under the eyes, most noticeable in the morning, dyspnea, cardiac palpitation, etc. Vomiting, without apparent cause, headache, vertigo, defective vision, and more or less generalized dropsy are also symptoms. Transient blindness is not uncommon in the early stages, but late in its course permanent loss of vision may occur, due to retinal disease. Hypertrophy of the heart, with high arterial tension and accentuation of the aortic second sound are common accompaniments. Bronchial catarrh, with edema of the larynx, may also develop. Anemia is pronounced on account of the large loss of albumin and gastro-intestinal disorders, and neuralgic pains are common. Uremic asthma and other manifestations or uremia may present themselves at any time. Irritability of the bladder is a very constant symptom, occurring very early in the course of the disease.

The Urine.—The urine is scanty, high-colored, albuminous, and under the microscope shows hyaline and granular tube-casts, granular epithelium, and if fatty degeneration occur, fatty tube casts and oil globules. The increase above the normal amount of the urine, as the disease progresses, must not be forgotten when the specific gravity is low, 1.010-1.015, and the quantity of albumin is increased. The normal constituents of the urine, and particularly urea are diminished. In the hemorrhagic form, blood is present in the urine.

Complications.—Uremia, edema of the lungs, pneumonia, pleurisy, pericarditis, peritonitis, meningitis, cardiac hypertrophy, and apoplexy are the most common complications.

Prognosis.—Complete recovery never occurs. Well-marked cases may terminate in death within a few months or two years while milder cases under appropriate treatment may be prolonged for an indefinite period. The appearance of complications and acute exacerbations are of unfavorable significance. The presence, persistently, of fatty tube-casts and oil globules in the urine is likewise unfavorable. I have seen four apparent recoveries, one after twelve months' duration, another after two years' duration, and still another after five years' duration, no return showing itself after two years. The secondary contraction of the kidneys must always be kept in mind, the particular symptoms of which are increased flow of urine of low specific gravity, with small amount of albumin, and hypertrophy of left ventricle, with accentuated aortic second sound. It is to be borne in mind that the course of a case of chronic Bright's disease is not continuously downward; periods of remission often follow the most aggravated symptoms, the patient and his friends, being buoyed with the hope of an early recovery, when, suddenly, an attack of acute uremia terminates life.

Treatment.—Rest is the first indication in the treatment. Residence in a dry and warm climate is a very useful adjunct. Woolen underclothing should be worn. The diet is of prime importance and should be so arranged as to reduce the quantity of nitrogenous foods to a minimum. It may consist of an absolute milk regimen, pure, or prepared as most palatable, or an exclusive lean meat diet, prepared by finely chopping, removing all fibrous and fatty portions, boiled quickly, salted to taste, and served hot—the so-called "Salisbury steaks." The use of half a pint of hot water, acidulated with lemon, before each meal is valuable.

The elimination of the effete matters, retained in the blood by reason of the crippled condition of the kidneys, may be brought about by catharsis, diuresis, and diaphoresis. The use of cathartics aids in reducing any existing edema and prevents to some

℞ Strych Sulph gr 1/30
Caffein Cit gr ij-V
Spartein Sulph gr 1/8-1/4
M. Ft Caps No I. Sig every 3-4 hours. } = Good Diuretic

extent the onset of uremia. The following is of value when the urine is scanty and the bowels are costive:

℞. Hydrargyri chlor. mitis,		
Pulv. scillæ,		
Pulv. digital	āā gr. j	āā .065 Gm.

M.—Ft. pil. No. i.

S.—To be taken three times daily.

Excessive dropsy will call for the administration of compound jalap powder, magnesium sulphate, elaterium, or the alkaline mineral waters combined with free diaphoresis. Diuretics are of value when the urine is scant, the most useful of which are digitalis, caffein citrate, spartein sulphate, nitroglycerin, potassium citrate, diuretin, and water. Dry cups or poultices over the loins and the injection of normal salt solution into the bowels or beneath the skin have diuretic properties. Diaphoresis may be brought about by the use of the warm bath, the Turkish bath, the warm pack, and vapor or hot-air bath together with friction and the administration of pilocarpin in some form. The following ointment is extremely valuable:

℞. Pilocarpinæ nitrat.	gr. j-iij	.065-2 Gm.
Petrolatum	℥ j	30. Gm.

M. S.—Apply piece size hickory-nut over dorso-lumbar regions, night and morning, covering surface with layer of cotton or gauze.

The anemia requires the administration of some preparation of iron, the best of which for this condition is Basham's mixture on account of its diuretic properties. The addition of the spirits of nitroglycerin adds to its efficacy. Cod-liver oil and arsenic are also of benefit. Drugs such as ergot, quinin, gallic acid, sodium benzoate, tincture of cantharides, and potassium iodid are believed to exercise an influence in checking the waste of albumin and are sometimes employed. The following formula has been employed for the anemia with gratifying results:

℞. Strychninæ sulph.	gr. ¼	.016 Gm.
Tinct. ferri chloridi	fʒ ij	8. Cc.
Acidi acetici puræ	fʒ j	4. Cc.
Curacoæ albæ	f℥ j	30. Cc.
Liq. ammonii acetat..ad	f℥ vj	180. Cc.

M. S.—Tablespoonful every five hours, followed by a glass of cold water.

Other Good diuretics = { Agurin
Theocin
Theobromine
diuretin. } = Average dose 5 gr every 4 hou

When the dropsy becomes marked the various measures already mentioned should be freely employed and often it will be necessary to resort to tapping and to multiple punctures of the skin. Uremia (*q. v.*) will demand special treatment.

Surgical Treatment.—Edebohls has proposed and employed decapsulation or decortication of the kidney in this disease with encouraging results. The operation consists in stripping off the capsule and cutting it entirely from the organ.

INTERSTITIAL NEPHRITIS.

Synonyms.—Chronic Bright's disease; sclerosis of the kidneys; contracted kidney; small red kidney; gouty kidney.

Definition.—An inflammation of the intervening connective-tissue of the kidney, chronic in its progress, resulting in an induration or hardening, with contraction of the organ; characterized by the frequent voiding of large amounts of pale, albuminous urine, of low specific gravity, disorders of the gastrointestinal canal and nervous system, and a strong tendency to cardiac hypertrophy and changes in the vessel. Albuminuria may be absent.

Causes.—The disease occurs usually in males from forty to sixty years of age. It may be primary, or secondary to chronic parenchymatous nephritis. Gout, chronic lead-poisoning, syphilis, alcoholism, opium habit, chronic cystitis, chronic gonorrhea, long-continued worry, anxiety or grief, alterations in the renal ganglionic centers, hereditary influences, passive congestion from heart-disease, and hepatic disorders are the principal causes.

Pathologic Anatomy.—Both kidneys are usually involved and their size is diminished. The capsule is thickened, opaque, and adherent. The surface of the kidney is granular, with cysts of various sizes, of transparent color, scattered irregularly over the surface. On section, the tissue of the kidney is tough and resistant. The cortical portion is thin, from atrophy, being only a line or two in thickness. The connective-tissue is greatly thickened, compressing the tubules into mere threads, the glomeruli being grouped together in bunches, owing to the wasting of the intermediate tubes. The color varies from a dark brown to a yellowish gray, according to the amount of blood in the organ.

The left side of the heart is hypertrophied, and there is also hypertrophy of the muscular fiber of the arterioles throughout the body; if the case is protracted, the hypertrophied tissues undergo fatty degeneration. Cardiac degeneration with arterio-capillary sclerosis or fibrosis is associated with advanced nephritis. The changes in the arterial walls lead to apoplexy, albuminuric retinitis, and fatty degeneration and atrophy of the ganglionic centers.

According to Tyson the kidney may be atrophic and possess an excess of connective-tissue as the result of senility, independent of chronic interstitial nephritis.

Symptoms.—The onset is insidious, and often marked alterations in the kidneys, heart, and vessels have occurred before the disease is recognized. There are no characteristic early symptoms in the majority of cases, the disease being apparently latent until some special outbreak causes a more thorough examination of the patient, when interstitial nephritis is detected.

Any of the following symptoms may first attract attention: Frequent micturition; increased amount of acid urine, fifty to ninety ounces, and of a pale color; low specific gravity, 1005-1015; containing a small amount of albumin, which may be absent for days; occasional epithelial cells and hyaline and pale granular casts. No dropsy, but a little puffiness and edema of the conjunctiva—the Bright's eye. Subconjunctival ecchymoses. Disorders of vision. Albuminuric retinitis. Forcible cardiac action with high arterial tension, due to left cardiac hypertrophy, which is an almost constant condition. Attacks of vertigo; headache; pulsations in the head, neck, and other parts of the body, and, as the disease progresses, cardiac distress, dyspnea, and palpitation occur. A reduplication of the first cardiac sound is common; the second aortic sound is accentuated and the pulse is hard and resisting, indicating high tension and thickening.

Progressive anemia is a frequent symptom. There is great weakness; the body weight declines; the skin is dry and scurfy; and there is shortness of breath on exertion. Albumin may be constantly absent from the urine, and casts be only occasionally detected after many trials and yet the disease will progress towards a fatal termination. Towards the end, the urine diminishes in quantity, the specific gravity increases, and the casts become more numerous and various, dark granular and blood casts often being observed.

Uremia may occur at any time and may be manifested by persistent dyspepsia, occasional vomiting, headache, vertigo, stupor, drowsiness, violent itching of the skin, tremors, convulsions, epileptic seizures, or apoplectic attacks. The duration is indefinite and the termination is usually in death by convulsions and coma.

Complications.—Bronchitis, pneumonia, pleurisy, pericarditis, cardiac hypertrophy, uremia, albuminuric retinitis, and apoplexy, are the most common complications.

Diagnosis.—Interstitial nephritis is most likely to be confounded with parenchymatous nephritis. The following table from Millard presents the most important points of difference between the two:

CHRONIC PARENCHYMATOUS NEPHRITIS,	CHRONIC INTERSTITIAL NEPHRITIS,
The urine is always albuminous.	Urine not constantly albuminous.
Urine usually scanty.	Urine usually abundant.
Dropsy and edema almost always occur.	Dropsy seldom or never present; sometimes slight edema.
Hypertrophy of the heart seldom exists.	Some hypertrophy of heart, with increased arterial tension, almost always present.
high early low later Specific gravity of urine usually higher than the normal. Urine darker and with less of a soapy appearance than in chronic interstitial nephritis.	Urine generally of a light color and low specific gravity.
Uremic symptoms less frequent than in chronic interstitial nephritis.	Uremic symptoms are met with in their most pronounced form, and usually occur in severe cases.
Epistaxis and cerebral hemorrhages rare.	Epistaxis and cerebral hemorrhages frequent.
Occurs most frequently before the age of forty.	Occurs most frequently after forty.
Blood corpuscles and connective-tissue shreds more frequently found in chronic croupous nephritis.	Absent in chronic interstitial nephritis.

Course is chronic with considerable anemia.	Development more gradual, the health of patient often less impaired, and duration longer than in chronic croupous nephritis.
Casts more numerous and in greater variety than in chronic interstitial nephritis; waxy, granular, fatty, and hyaline casts occurring.	Casts rare, the hyaline variety being most frequently encountered.
Epithelia from the kidney and pus corpuscles more numerous than in interstitial nephritis.	Kidney epithelia and pus corpuscles scanty, and occasionally absent.
Urates and phosphates predominate; oxalates rare.	Oxalate of lime almost always occurs.
Albuminuric retinitis rare.	Albuminuric retinitis common.
Gangrenous erysipelas and phlegmonous swellings more common; also dyspepsia and anemia.	
Visceral complications, as pneumonia, pleuritis, pericarditis, and bronchitis, not uncommon.	Visceral complications rare.
Diarrhea occasionally.	
Cirrhosis of liver rare.	Cirrhosis the most frequent hepatic lesion.
Atheroma of arteries rare.	Atheroma common.

Prognosis.—Recovery never occurs. The disease is essentially chronic; cases have lasted as long as eleven years. Uremic symptoms are of unfavorable significance.

Treatment.—The diet should be carefully regulated and nitrogenous foods should be eliminated. Milk, (plain, skimmed, or diluted with Vichy), eggs, (soft-boiled or poached in milk), chicken broth, and vegetables should constitute the larger portion of the food. Alcoholic stimulants should be avoided. Physical and mental rest should be advised. A daily warm or hot bath is valuable but under no consideration should cold or sea-bathing be allowed. Warm clothing should be worn and the body should be protected from cold and dampness. Regularity in the bowel movements is desirable and for this purpose the alkaline mineral waters, the salines, or cascara sagrada should be administered. Iron will be necessary to combat the anemia and potassium iodid may be of value in lessening the connective-tissue hypertrophy. Headache, vertigo, and similar symptoms dependent upon increased arterial tension may be relieved by the use of spirits of nitroglycerin, ♏i (0.06 Cc.), or nitroglycerin, gr. 1-100 (0.00065 Gm.), three times daily. Opium or any of its preparations should never be employed. When a hypnotic is required sulfonal, trional, or paraldehyde should be used. In the early stages of the disease the following formula will be found very valuable:

℞. Hydrargyri chloridi corrosiv..	gr. j	.065 Gm.
Aurii et sodii chloridi........	gr. j	.065 Gm.
Ferri reduct.	gr. xxx	2. Gm.
Spts. glonoini...............	♏xxx	2. Cc.

M.—Ft. pil. No. xxx.
S.—One after meals.

For gastric symptoms the following is an excellent formula:

℞. Pepsin. puræ............	gr. xxxij	2. Gm.
Acidi hydrochloric. dil. .	f℥ ss	15. Cc.
Glycerini	f℥ j	30. Cc.
Aquæ chloroformi ..ad	f℥ iij	90. Cc.

M. S.—One teaspoonful at meal-time, well-diluted.

AMYLOID KIDNEY.

Synonyms.—Chronic Bright's disease; waxy kidney; lardaceous kidney.

Definition.—A peculiar infiltration into, or a degeneration of, the structure of the kidney, from the deposit of an albuminoid material, having a superficial resemblance to molten wax or boiled starch. Similar changes occur in the liver, spleen, intestines, and other organs.

Causes.—The chief cause is prolonged suppuration, especially of the bones; coxalgia; syphilis; cancer; phthisis.

Pathologic Anatomy.—The kidney is uniformly enlarged. It presents a pale, glistening, translucent appearance, and has a doughy consistency. On section, the surface is homogeneous, anemic, and whitish. The deposit occurs along the renal vessels and in the vascular tufts of the glomeruli, progressing until all parts of the organ are infiltrated. When the organ is thus infiltrated, the structure proper undergoes an atrophic degeneration, the result of pressure.

The reaction with iodin and sulphuric acid affords a certain test for the amyloid deposit. Brush over a section of the affected kidney a solution of iodin with iodid of potassium in water, when a mahogany color will be produced, and if diluted sulphuric acid is now added a violet or bluish tint results. A very pretty reaction is made by contact with a one per cent solution of anilin violet, which strikes a red or pink color with the amyloid material, while the unaltered tissues are stained blue, making a beautiful contrast.

Similar changes occur in other organs of the body. With the amyloid change may be associated either parenchymatous or interstitial nephritis.

Symptoms.—Together with the symptoms of the underlying causes there are edema of the lower extremities, ascites, increased flow of pale watery urine of low specific gravity containing albumin and hyaline and waxy tube-casts, and sometimes diarrhea. The liver and spleen are enlarged. Uremia, cardiac hypertrophy, or increased arterial tension are extremely rare in this disease unless other forms of nephritis are present coincidently.

Diagnosis.—The history of prolonged suppuration, the enlargement of the liver and spleen, and the increased flow of pale urine containing waxy casts which give the amyloid reaction serve to distinguish this disease from other renal affections.

Prognosis.—If the underlying disease can be cured before the amyloid change has been fully developed it may be arrested; otherwise a fatal termination may be expected in from a few months to a year.

Treatment.—In addition to measures directed towards the primary cause, every effort should be made to sustain the patient. For this purpose a generous diet, syrup of iodid of iron, cod-liver oil, quinin, ammonium chlorid, etc., should be freely administered. In cases due to syphilis, potassium iodid and small doses of bichlorid of mercury, gr. 1-50 to 1-35 (0.0015-0.002 Gm.), should be given over an extended period.

PYELITIS.

Synonyms.—Suppurative nephritis; pyelo-nephritis.

Definition.—An acute catarrhal inflammation of the pelvis of the kidney; the term *pyelo-nephritis* is used when suppurative inflammation is superadded to the catarrhal inflammation. The disease is characterized by lumbar pains, irritability of the bladder, the urine being neutral or alkaline in reaction and milky in appearance; if pyelo-nephritis occurs symptoms of hectic fever and exhaustion are added, the urine containing pus.

Causes.—Cold or exposure; cystitis; obstruction of the ureters by renal calculi; pressure from a tumor; prolonged use of bromids and other irritative drugs; rheumatism; and as a sequel of infectious diseases.

Pathologic Anatomy.—The inflammation is at first catarrhal; it is characterized by injection of the mucous membrane of the pelvis of the kidney, with slight extravasations of blood; relaxation and softening, shedding of the epithelium, and the subsequent discharge of mucus and pus. If the morbid condition has existed for some time, the kidneys, one or both, are in a process of suppuration; they are enlarged, deeply congested, except where suppuration is proceeding, when they are of a yellowish-white color—*pyelo-nephritis*. Pus is constantly forming, and if there be no obstruction, flows away with the urine; should there be an impediment to its escape, pus accumulates in the pelvis of the kidney, causing its distention, giving rise to the condition known as *pyelo-nephrosis*. The pressure caused by the obstruction finally leads to destruction of the entire organ, a mere sac, or renal cyst, remaining.

Symptoms.—The affection begins with chilliness, feverishness, lumbar pains following the course of the ureters, and frequent or neutral in reaction and deposits a copious whitish or yellowish-white sediment containing a small amount of albumin. Blood will be present if the condition is due to a renal calculus. The formation of pus is indicated by chills, irregular fever, sweats, localized pain, enlargement, and tenderness in lumbar region, the presence micturition. The urine is milky in appearance when voided, acid of pus in the urine, and leukocytosis. In marked cases there are low muttering delirium, fissured and dry tongue, anemia, emaciation, stupor, and coma. If both kidneys are involved uremia may supervene.

Diagnosis.—*Cystitis* may be distinguished by its history, the absence of lumbar pains, and the alkaline urine.

Perinephritic abscess or suppuration of the loose cellular tissue surrounding the kidney, is characterized by localized pain, swelling, tenderness, and edema in the lumbar region with chills, fever, and sweat, but the urine remains normal.

Renal calculus may give rise to pyelitis and in such cases renal colic, the passage of the stone, and the presence of blood in the urine will aid in making the diagnosis.

Tuberculosis of the pelvis of the kidney has many points in common with simple pyelitis, but in the former there are in addition tuberculous foci elsewhere in the body and tubercle bacilli may be found in the urine.

Prognosis.—Simple catarrhal cases in which there is no obstruction to the discharge recover usually in a week or ten days. In the presence of an obstruction or suppuration the prognosis is unfavorable.

Treatment.—Rest in bed and a milk diet are essential. Free diaphoresis and the free consumption of water to dilute the urine are indicated. The alkali waters such as Buffalo Lithia Springs water and the Rockbridge Alum Springs water of Virginia are valuable. Local applications of heat to the lumbar region and use of opium will be required to relieve the pain. The character of the renal secretion may be altered by the administration of tar, santal wood oil, copaiba, eucalyptol, turpentine, cubebs, benzoic acid, salol, or urotropin. If there is renal hemorrhage alum, gr. xx (1.3 Gm.), may be used. The rapid exhaustion calls for the use of tonics, particularly quinin, strychnin, iron, etc. As suppuration is likely to supervene at any time, a surgeon should be consulted early as a prompt operation may be the means of saving otherwise hopeless cases.

RENAL CALCULI.

Synonyms.—Nephro-lithiasis; gravel; renal colic.

Definition.—Renal calculi are concretions formed by the precipitation of certain substances from the urine, around some body or substance acting as a nucleus.

Their presence may not be recognized until one or more attempt to pass along the ureters, when an attack of renal colic results; or, by irritation pyelitis is produced; or, more rarely, they are voided by the urine without exciting any symptoms. By *gravel* is meant very small concretions (sand), which are often passed in the urine in large numbers.

Causes.—The affection occurs at all ages, but is most common in individuals from forty to fifty years of age. A special liability seems to exist in some families but the precise etiology of renal calculi is as yet undetermined.

Characteristics.—In structure a urinary calculus consists of a central nucleus surrounded by a body, outside of which there may be a phosphatic crust. The nucleus may or may not be of the same material as the rest of the stone, sometimes being a foreign body, or inspissated mucus or blood. On section the stone shows a stratified arrangement, often radiated. They occur in several varieties:

1. *Uric acid,* as calculi and gravel, and especially associated with the gouty diathesis.

2. *Urates,* chiefly urate of ammonium; nearly always in childhood.

3. *Oxalate of lime* or mulberry calculus; characterized by hardness, roughness, and very dark color.

4. *Phosphatic calculi* form as frequently in the bladder as in the kidney, and present a chalky or earthy appearance.

5. *Alternating calculi,* consisting of alternate layers of two or more primary deposits.

Symptoms.—In the absence of renal colic there are usually no symptoms to attract the attention. Renal colic is manifested motion, attended by retraction of the testicle on the corresponding chiefly by agonizing pain in the back, principally in the dorsolumbar region, which radiates along the ureters and is worse on side, irritability of the bladder, pallor of the face, pinched features, nausea, vomiting, lowering of surface temperature, faintness, and rarely unconsciousness. The paroxysm terminates suddenly after some minutes or a few hours, the stone escaping into the bladder. If the stone is not passed the attack may subside to recur within a short period. The urine is more or less suppressed usually escaping in drops and stained with blood. If the condition is bilateral and both ureters are obstructed, uremic symptoms occur. This is rare. Obstruction of the ureter by a calculus, if unrelieved may terminate in pyelitis, hydronephrosis, or pyonephrosis. Suppuration is indicated by chills, irregular fever, sweats, and leukocytosis.

During the interval between the attacks there are more or less pain and tenderness over the region of the kidneys and the urine is stained with blood. Its specific gravity is high, and albumin and long, narrow, hyaline casts are present. Epithelium from the pelvis of the kidney, pus, and crystals indicating the character of the calculus may also be found.

Diagnosis.—The distinctive features of this affection are pain and tenderness in the back, persistent hematuria, albuminuria, scanty urine of high specific gravity, containing hyaline casts, pus, and crystals, and the characteristic paroxysms of renal colic. The X-ray should be employed in all cases to confirm the diagnosis.

Prognosis.—The outlook is guardedly favorable in the absence of complications. Impaction may produce extensive disorganization of the kidneys, or its passage along the ureter may prove fatal. Recurrences are common. The condition known as gravel is the least dangerous. If the stone is large or there are more than one, the prognosis becomes correspondingly more serious.

Treatment.—During the attack, a hot bath should be ordered and a hypodermic injection of morphin and atropin, or a suppository of extract of opium, gr. i (0.065 Gm.), and alcoholic extract of belladonna, gr. ss (0.032 Gm.), should be administered. Hot poultices and hot fomentations should be applied to the lumbar region, and diluent drinks freely consumed. Chloroform may be necessary to relieve the pain in some cases. The coal-tar products are of value at times. During mild attacks of gravel, solution of potassium citrate, f℥ss (15 Cc.), alone or combined with camphorated tincture of opium, fʒss (2 Cc.), is of value. Hematuria may be relieved by alum, gr. xx (1.3 Gm.), or:

℞.	Extracti ergotæ fluidi,	
	Tinct. krameriæ......āā f℥ ii	āā 60 Cc.

M. S.—One teaspoonful every two hours.

When the calculi are large, numerous, and impacted, or threaten life, a surgical operation should be performed for their removal.

During the interval efforts should be made to prevent the formation of the calculi. There are no remedies that will dissolve fully-formed calculi, but there are many methods by which the various crystals in the blood and urine may be kept in solution and thus prevent the formation of concretions. If repeated examinations of the urine show a tendency towards the uric acid diathesis, the alkalies, such as Buffalo Lithia Springs, Rockbridge Alum Springs, Saratoga, Vichy, Bedford, Poland, and similar waters, potassium tartraborates (cream of tartar, four parts, boric acid, one part, water, ten parts; dose, gr. xx three times daily diluted), lithium citrate, gr. v-x (0.3-0.6 Gm.), or the following should be administered:

℞.	Magnesii carbonat. ......ʒ j	4. Gm.
	Acid. citrici ....ʒ ij	8. Gm.
	Sodii borat. ....ʒ ij	8. Gm.
	Aquæ bullientis ....℥ viij	240. Cc.

M. S.—Tablespoonful three times daily, diluted.

The diet in these cases should consist largely of milk and vegetables, using only a very small quantity of meat and other nitrogenous foods.

If there is a tendency towards the deposition of phosphates with the formation of calculi, a diet of meat and nitrogenous substances, acidulated drinks, distilled water, benzoic acid, and boric acid, are indicated.

Either form of treatment will be of equal value if there is any tendency towards concretion of the oxalates. Piperazin, gr. v (0.32 Gm.), three times daily, has been employed with success in renal calculi.

HYDRONEPHROSIS.

Definition.—A cystic condition of the kidney, due to retention of secretion. It may be due to impaction of a stone in the ureter, stenosis or congenital stricture of the ureter, or some morbid growth.

The affection begins with obstruction of the ureter, and is followed by dilatation of the pelvis of the kidney. As the fluid accumulates it presses on the parenchyma and induces gradual wasting of that structure.

Symptoms.—When slight, there are no distinctive manifestations, but when the amount of fluid is large, there appears in the lumbar region a soft, fluctuating, painless tumor, over which dullness may be obtained by percussion. A clear fluid, containing urea and uric acid, will be withdrawn on aspiration. The condition may be intermittent or constant, according to whether the obstruction is or is not relieved.

Diagnosis.—The history, gradual onset, location of the tumor, the relation of its size to the urinary excretion, and the character of the aspirated fluid will aid in making a diagnosis, but often an exploratory incision is necessary.

Prognosis.—The affection is serious, in that if unrelieved, it tends towards distintegration of the kidney substance, pyonephrosis, rupture, and ultimately death.

Treatment.—The treatment is entirely surgical and includes aspiration, nephrotomy, and nephrectomy.

TUBERCULOSIS OF THE KIDNEY.

Tuberculosis of the kidney is seldom a primary affection, and is usually a part of a tuberculous infection involving the entire urinary tract. It is most common in young adult males, and its etiology is that of tuberculosis in other regions. As elsewhere, its lesions may be military tubercles or caseous nodules. The symptoms resemble pyelitis from other causes, and a distinction often can only be made by the detection of tubercle bacilli in the urine. The affection is very grave, and there is no satisfactory medical treatment. A surgical operation offers the only hope of relief. Untreated cases live from a few months to two or three years.

PERINEPHRIC ABSCESS OR PARANEPHRITIS.

Inflammation of the connective-tissue surrounding the kidney terminating in suppuration and abscess formation. It may arise from traumatism, or it may be due to extension by perforation of

Sudden uraemic coma is more common
chronic interstitial nephritis. (Osler)

a renal or other abdominal abscess. It is attended by localized pain, tenderness, and edema, and patient flexes the corresponding thigh when sitting or lying to afford more comfort. Chill, fever, and sweats are present. The condition is surgical in character, but should always be considered as a possibility in medical renal affections attended by hectic symptoms.

ACUTE UREMIA.

Synonyms.—Uremic poisoning; uremic intoxication; uremic coma; uremic convulsions.

Definition.—A group of nervous phenomena, which may develop during the course of acute or chronic Bright's disease, and other maladies, the result of the retention or accumulation in the blood of an excrementitious material, supposed to be urea. Not due to ure

Causes.—Suppression of urine, from acute or chronic Bright's ✗ disease, probably more frequent in chronic parenchymatous nephritis; cystic, tubercular, or cancerous kidney; the puerperal state; operations on the uterus bladder, urethra, or rectum.

Symptoms.—Uremic intoxication is the result of the failure of the kidneys to perform their normal function of eliminating some or all of the poisonous elements of the urine.

The toxemia may develop suddenly, by a convulsive seizure followed by coma, or slowly and gradually. Usually the attack is preceded by a decrease in the urinary secretion and slight or marked edema in various parts of the body; although it must be borne in mind that in rare instances, during, or immediately prior to, the appearance of the uremic phenomena, the normal urinary flow has been largely exceeded.

The acute outbreak may manifest itself in a variety of ways.

Gastrointestinal variety: The patient suddenly experiences attacks of vertigo, pallor of face, nausea and vomiting, with fever, the temperature varying between 100° and 103°, pulse tense and rapid, respiration hurried, and the urine scanty with low specific gravity; unless symptoms are promptly relieved, convulsions or drowsiness may occur, followed by coma and death. Rarely an acute maniacal outbreaks follows the gastrointestinal symptoms.

Convulsive variety: Without any appreciable prodromes, epileptiform convulsions, may occur, with or without loss of consciousness.

The convulsions may consist of a single paroxysm, or a succession of fits may follow one another at intervals of a few minutes or several hours, the patient being in a condition of more or less profound insensibility during the intervals. The fits closely simulate true epilepsy. In this variety the temperature is high, from 103° to 106° or more, the pulse rapid, with or without tension, the respirations quickened. Coma followed by death is a very common ending of this variety of uremia, or after a profound sleep of hours the patient gradually recovers his usual health. Alcoholic excesses are responsible for many of these attacks.

Cerebral variety, or *uremic coma:* Develops gradually, with an increasing drowsiness, associated with headache and irritability of temper (mild mania). Nausea, vomiting, and rise of temperature, often reaching 105°, rarely 107°, with rapid, full pulse may be present, or the patient may fall suddenly into a condition of profound coma, the symptoms closely resembling an apoplectic stroke, excepting the high temperature. Temporary blindness and transient paralysis are not uncommon. Uremic coma is always accompanied with rise of temperature and stertor. "The stertor is peculiar; it is not the 'snoring' of apoplexy, but a sharp, hissing sound produced by the rush of expired air against the teeth or hard palate." (Loomis.) The respirations are accelerated, the pulse rapid but minus tension. This variety may suddenly terminate fatally with a convulsion, or a deepening coma with prostration and cold, wet skin, with edema of the lungs, or, rarely, in gradual recovery.

Diagnosis.—Uremia resembles a number of conditions in which convulsions and coma are prominent symptoms. A specimen of urine should always be obtained and tested for albumin. The quantity is scant and the percentage of urea is decreased. Other signs of kidney disease are present. The breath has a urinous odor; the arterial tension is often high; the second aortic sound is accentuated; the pupils are small and equal, and in acute outbreaks there is a rise of temperature due to irritation of the heat centers.

Uremic coma may closely simulate coma from other causes. *Cerebral apoplexy* may be distinguished by its history, the age at which it occurs, generalized arterial sclerosis, the slow noisy irregular respiration, the pulse is slow and full, the pupils are uninfluenced by light, conjugate deviation of the eyes, the face is flushed, subnormal temperature at first, with a subsequent rise above normal, permanent one-sided paralysis, and an absence of the urinary symptoms common to uremia.

Epilepsy is attended by coma of short duration. The attack is preceded by a sharp cry and extreme pallor of the face, the countenance being dusky in uremia. The history, age of the patient, and the presence or absence of urinary symptoms should be considered in making the diagnosis.

Sunstroke may be recognized by the accompanying circumstances, the history, the extremely high temperature, and the absence of albuminuria, and other characteristic urinary symptoms of uremia.

Opium poisoning is manifested by contraction of the pupils, slow respiration, slow full pulse, and the odor of laudanum at times. An examination of the urine will exclude uremia.

Alcoholism may be differentiated by its history, odor of the breath, incomplete loss of consciousness, and the absence of urinary symptoms, dropsy, etc. Pressure applied over the supraorbital notches with increasing force will serve to produce consciousness in alcoholism.

Prognosis.—The condition is very serious. The attack may end in recovery, but recurrences are common and the affection ultimately terminates in death.

Treatment.—During an attack, while the patient is unconscious, elimination should be procured by the administration of one or two drops of croton oil, diluted by glycerin or sweet oil, or a quarter of a grain (0.0165 Gm.) of elaterium in solution by the mouth. The following enema may be used:

℞.	Magnesii sulph. ....℥ ij	60. Gm.
	Glycerini ....℥ j	30. Cc.
	Aquæ bul. ....℥ iv	120. Cc.

M. S.—As enema.

Free sweating should be encouraged by the use of the hot pack, vapor bath, or hot-air bath, together with the hypodermic injection of pilocarpin hydrochlorate, gr. ¼ (0.016 Gm.), or the rectal injection of an infusion of jaborandi leaves (jaborandi ʒi, water ℥iv). In robust individuals, venesection, or cupping, may be performed. In feeble patients, during diaphoresis, the tendency towards edema of the lungs should be combatted by the hypodermic injection of atropin sulphate, gr. 1-60 (0.001 Gm.), and strychnin sulphate, gr. 1-32 (0.002 Gm.). The convulsions are relieved by inhalations of chloroform, chloral by the rectum, morphin hypodermically, and venesection. When the flow from a vein is only a few drops, it may be increased by the hypodermic injection of amyl nitrite, ♏ v (0.3 Cc.), with aromatic spirits of ammonia, ♏ xv (1 Cc.).

Diuresis should be promoted by dry or wet cupping, poultices over the loins, hot compresses of infusion of digitalis over the abdomen, pilocarpus rubbed over the kidneys, and the use of normal salt solution subcutaneously or by the bowel. Drugs such as infusion of digitalis, citrate of caffein, spartein sulphate, nitroglycerin, and diuretin may be given hypodermically, or by the mouth if the patient is able to swallow.

During the intervals or the prodromal period, these measures should also be employed together with other drugs by the mouth. The diet should be milk in large quantities. Sodium benzoate, ʒ i-ii (4-8 Gm.), in twenty-four hours, may be administered as it is believed to materially influence the condition.

When the gastrointestinal variety is present the patient should be placed in bed and the magnesium sulphate enema and caffein citrate, gr. iii (0.2 Gm.), every three hours, should be administered. When the secretions have started one of the following powders should be given every two hours for twenty-four hours followed by Hunyadi Janos water:

℞.	Hydrargyri chlor. mitis..gr. ¼-½	.016-.032	Gm.
	Sodii bicarb.............gr. ij	.130	Gm.
	Pulv. ipecacuanhæ.......gr. ⅙	.011	Gm.

M.—Disp. in chart. No. i.
S.—Use as directed.

The following formulas will be found of great value in bringing about diaphoresis:

℞.	Sparteinæ sulphat. ......gr. iv	.265	Gm.
	Pilocarpinæ hydrochlor. .gr. j	.065	Gm.
	Infus. digital. ....f℥ ij	60.	Cc.

M. S.—Teaspoonful every half hour or hour until desired effect is obtained.

Or—

℞.	Digitalinæ cryst.	gr. 1-64	.001 Gm.
	Pilocarpinæ hydrochlor. .	gr. ¼	.016 Gm.
	Sparteinæ sulph.	gr. ½	.032 Gm.
	Aquæ destil.	♏xv	1. Cc.

M. S.—For hypodermic use; to be repeated as necessary.

Or—

℞.	Pilocarpinæ nitrat.	gr. ij	.13 Gm.
	Petrolat.	f℥ j	32. Gm.

M. S.—Apply locally over the kidneys twice daily.

MOVABLE KIDNEY.

Synonyms.—Floating kidney; wandering kidney; nephroptosis.

Definition.—A condition of the kidney, either congenital or acquired, in which the tissues around about the organ are so lax and the renal vessels so elongated as to permit the kidney to be moved in certain directions, causing a movable tumor in the abdomen.

Causes.—The kidney is normally held in position by the layer of peritoneum which is attached to the anterior surface of its adipose capsule. In movable kidney the adipose tissue, in which the normal kidney is imbedded, disappears. The renal vessels are in many cases abnormally long. The condition may be congenital or acquired and occurs with greatest frequency in women. Relaxation of the abdominal walls from pregnancy or other conditions, the wearing of tight corsets or girdles about the waist, violence, increased weight of the kidney, pressure of adjacent tumors, traction of hernia, and rapid emaciation are important etiologic factors.

Symptoms.—Subjective symptoms may be absent entirely. In many cases, the patient experiences a heavy, dragging pain in the abdomen, aggravated by standing or walking. Gastrointestinal symptoms, mental anxiety, and hysterical manifestations, often accompany the condition. Various reflex disturbances may be present such as palpitation of the heart, neuralgic pains, cardialgia, irritability of the bladder, etc. Torsion of the ureter and renal vessels may occur at any time and is manifested by paroxysms of intense pain with symptoms of collapse. The kidney at times swells without apparent cause and becomes sensitive to the touch. Its tendency to change its position is its characteristic feature and the displaced organ may be found anywhere in the abdomen. Gastroptosis or enteroptosis may accompany it.

Diagnosis.—Physical examination is necessary in all cases. The detection of a freely movable reniform tumor of fixed size and the absence of the kidney from its normal situation are the distinctive features. The right kidney is most often affected and is usually to be found somewhere in hypochondriac region. Pulsation of the renal artery may occasionally be felt.

Prognosis.—The affection is extremely chronic, but rarely, if ever, terminates fatally in the absence of complications.

Treatment.—Measures directed towards improvement of the general health are advised. In many cases the symptoms are entirely relieved by lying on the back or by wearing a suitable abdominal supporter to retain the kidney in its proper situation. When the paroxysms occur, rest in bed, hot applications over the lumbar regions, and opiates will be necessary. If these recur frequently, some form of surgical treatment such as nephrorrhaphy, fixing the organ by sutures, or extirpation will be required.

CYSTITIS.

Synonym.—Catarrh of the bladder.

Definition.—A catarrhal, croupous, or diphtheritic inflammation of the vesical mucous membrane, acute or chronic in course, characterized by rigors, moderate fever, hypogastric pain, frequent but scanty urination, pus in the urine (pyuria), and severe vesical tenesmus.

Causes.—*Acute cystitis* may be due to long-continued retention of urine, foreign bodies in the bladder, pyelitis, urethritis, myelitis, traumatism, or the infectious fevers especially diphtheria. *Chronic cystitis* may follow the acute variety or may arise from chronic Bright's disease, gout, calculi, or retention of urine such as follows enlarged prostrate and urethral stricture.

Pathologic Anatomy.—*Acute catarrhal cystitis* begins with hyperemia of the mucous membrane which is manifested by redness, swelling, and edema. If the congestion is intense, the smaller capillaries may rupture causing extravasation of blood. Following the hyperemia, increased secretion of the small glands at the base of the bladder and an increased growth and consequent desquamation of the vesical epithelium occur. If the inflammation is intense it may terminate in suppuration, ulceration, or in the formation of a false membrane.

In *chronic cystitis* "the mucous membrane is thick, blue-gray in color, and very tough. Muco-pus and viscid mucus are formed in large quantities upon its surface. The muscular wall of the bladder may sometimes be half an inch thick, and the fasciculi give a ribbed appearance to the internal surface, called the 'columnar bladder.' The hypertrophy of chronic cystitis may be eccentric or concentric. In some cases diverticuli are formed, in whose walls are dilated and tortuous veins. In nearly all cases bacteria are found in abundance." (Loomis).

Symptoms.—*Acute cystitis* is characterized by an abrupt onset with rigors, slight fever, loss of appetite, sleeplessness, and a feeling of depression. Micturition is frequent but the urine is only voided drop by drop and its passage is followed by distressing vesical tenesmus. Dull pain over the bladder and in the iliac regions, and burning along the urethra are present. The urine is cloudy, of an alkaline reaction, and at times fetid. Microscopic examination shows epithelium, pus, red blood corpuscles, and various forms of bacteria.

Chronic cystitis is attended by an insidious onset and is manifested by dull pain, and frequent, scanty urination. If there is ulceration of the vesical mucous membrane, severe localized pain, hematuria and emaciation will also be present. In all cases there are in addition the symptoms of some obstructive condition such as stricture, calculus, or enlarged prostrate together with debility and mental depression. The urine is alkaline and contains large amounts of muco-pus or pus. On standing it deposits a thick, glairy, viscid sediment in which triple phosphates and large pus corpuscles may be detected by the microscope. Although the quantity of urine voided by the patient is small, the use of the catheter after micturition will in most cases serve to withdraw several ounces of fetid, cloudy, alkaline urine.

Diagnosis.—The reaction and characteristics of the urine, together with the history will serve to distinguish cystitis from pyelitis, interstital nephritis, and similar conditions.

Prognosis.—The outlook in acute cystitis is, as a rule, favorable, but is controlled to a great extent by the character of the cause. The chronic variety tends to persist indefinitely and is incurable after hypertrophy of the bladder has occurred.

Treatment.—In the *acute variety*, the patient should be placed in bed, and a liquid diet, preferably milk, should be ordered being careful to eliminate all highly-seasoned articles. Warm applications should be applied over the bladder and occasionally cupping or leeching may be required. The urine should be well-diluted by large draughts of pure water or the alkaline mineral waters such as Farmville lithia, Buffalo lithia, Rockbridge alum, or Vichy waters. Alkalinity of the urine from any cause is relieved by the administration of ammonium benzoate, gr. xx (1.3 Gm.), in water or the solution of potassium citrate, ʒ i (3.7 Cc.). For the pain and tenesmus, a suppository of extract of opium and extract of belladonna may be necessary in addition to the hot applications and hot enemas. Fluid extract of cannabis indica, ♏ xv-xxx (1-2 Cc.) every three hours, often relieves the tenesmus. A free movement of the bowels obtained by the administration of a saline cathartic is always of value in lessening the inflammation and its attendant symptoms. The following formulas are also of decided value in this condition:

℞.	Acidi benzoici		
	Sodii borat.	āā ʒ ij	āā 8. Gm.
	Infusi buchu, vel		
	Infusi uvæ ursi	f℥ vj	180. Cc.

M. S.—Tablespoonful every two hours, well-diluted.

Or—

℞.	Tinct. hyoscyami	fʒ vj	24. Cc.
	Tinct. opii camph.	fʒ vj	24. Cc.
	Potassii bromidi		
	Sodii bicarb.	āā ℈ viij	āā 10.5 Gm.
	Liq. potassii citrat. .	q. s. f℥ viij	q. s. 240. Cc.

M. S.—One teaspoonful every two hours, well-diluted.

A valuable prescription is—

℞.	Ext. pichi fld.	f℥ j	30. Cc.
	Potassii nitrat.	ʒ j	4. Gm.
	Elix. simplicis	f℥ iij	90. Cc.

M. S.—One teaspoonful every two hours, well diluted.

Or—

℞. Liq. potassæ (B. Ph.)	..fʒ ij	8. Cc.
Ol. santal. flav.	........fʒ ij	8. Cc.
Aq. cinnamon	......ad f℥ viij	240. Cc.

M. S.—Tablespoonful three times daily, diluted. (Saundby).

Chronic cystitis requires also a mild unirritating diet and the free use of the alkaline mineral waters. The bladder should be emptied several times daily to prevent accumulation and consequent decomposition of the urine, the underlying cause meanwhile receiving appropriate treatment. Eucalyptol, gtt. x-xv (0.6-1 Cc.) every four hours diluted, fluid extract of grindelia, ♏ xx-fʒ i (1.3-4 Cc.), three times daily, or santal oil, gtt. v-x (0.3-0.6 Cc.), in emulsion or capsule after meals, may be administered internally. Urotropin, gr. v-viiss (0.3-0.5 Gm.), boric acid, gr. v-x (0.32-0.65 Gm.), benzoic acid, gr. v-x (0.32-0.65 Gm.), naphthalin, gr. ii (0.13 Gm.), salol, gr. x (0.65 Gm.), or resorcin, gr. v (0.32 Gm.) may also be employed. Irrigation of the bladder under aseptic precautions is a very important feature of the treatment. Tepid water should be used at first after which medicated solutions may be employed. Not more than from two to four ounces of fluid should be injected at first until the capacity of the bladder has been ascertained. Daily injections are usually sufficient. Sodium salicylate, ʒ i (4 Gm.), to the pint (½ liter), boric acid, ʒ i (4 Gm.), to the pint (½ liter), silver nitrate, gr. ¼ (0.016 Gm.) to the ounce (30 Cc.), or the following are the fluids most commonly used for this purpose:

℞. Sodii borat.	............℥ j	30. Gm.
Glycerini	.............f℥ ij	60. Cc.
Aquæ	................f℥ ij	60. Cc.

M. S.—Add one to two tablespoonfuls to warm water and use as directed.

DISEASES OF THE BLOOD.

EXAMINATION OF THE BLOOD.

Normal blood consists of corpuscles, plaques, and serum. The corpuscles are red and white. The ordinary red blood cell is 1-3200 of an inch in diameter and varies in number from 4,500,000 to 5,000,000 to the cubic millimeter. They contain hemoglobin, the oxygen carrier of the blood. The white blood cells measure 1-2500 of an inch in diameter and number from 7,000 to 10,000 to the cubic millimeter. The blood plaques number about 200,000 to the cubic millimeter.

The specific gravity of normal blood varies from 1050 to 1060. It may be ascertained by the preparation of a number of solutions of glycerin and water of varying specific gravities from 1040 to 1080. A drop of blood is placed in each solution. The solution in which the drop of blood remains stationary is of the same specific gravity as the blood under examination. Hammerschlag adds a drop of blood to a mixture of chloroform and benzol and then increases the quantity of either constituent until the drop of blood neither rises or falls but becomes stationary. The specific gravity of the mixture is then taken by the ordinary means. Increased specific gravity of the blood is observed in infancy and in acute febrile diseases such as diphtheria, pneumonia, pleurisy, etc. Decreased specific gravity is common in healthy women, and in anemia, chlorosis, and leukemia.

The reaction of the blood is normally alkaline, one cubic centimeter of blood being equal in neutralizing properties to 0.33 to 0.38 Gm. of sodium hydrate. This may be determined by titrating with a standard solution of acetic acid until it produces a change of color in contact with neutral litmus. Landois' method requires the preparation of a series of tartaric acid solutions beginning with a decinormal solution (7.5 Gm. tartaric acid to one liter of distilled water). One cubic centimeter of each solution is then mixed with 0.1 Cc. of blood in a watch glass and tested with litmus paper. The mixture in which the reaction is neutral should be noted. In the calculation it should be remembered that 0.4 Cc. of tartaric acid requires 0.0016 Gm. caustic soda for its neutralization. The alkalinity of the blood is diminished in pernicious anemia, simple anemia, leukemia, uremia, diabetes, jaundice, chronic rheumatism, gout, carbon dioxid and phosphorous poisoning, febrile affections, and cachectic conditions. It is said, by some observers, to be increased in chlorosis.

Coloration of the blood may vary considerably. To the unaided eye arterial blood appears bright red while venous blood is darker in color. Deficient oxidation from any cause gives rise to darkening of the arterial blood. The blood is pale in chlorosis, hydremia, and leukemia and is of an abnormally bright red color in poisoning by carbon monoxid. It assumes a brownish-red or chocolate color in poisoning by hydrocyanic acid, nitrobenzol, anilin, and chlorate of potash.

Hemoglobin, the coloring matter of the blood, may be approximately estimated by means of Fleischl's or Gower's hemoglobinometer, or by the specific gravity method. Fleischl's method requires a metal stand with a stage perforated by a central circular opening beneath which is placed a plaster of Paris reflector. A small cell having a glass bottom and divided into two compartments is provided to fit into the circular opening. A wedge-shaped piece of glass, colored with Cassius' gold-purple which increases in intensity as the thick portion is reached, is mounted in a frame and interposed between the reflector and the circular opening. A graduated scale is provided on the frame which may be moved back and forth by a rack and pinion. The wedge of glass is so situated as to obstruct only one-half of the area of the circular opening. In using the apparatus each compartment of the cylindric cell is filled with distilled water and the wedge-shaped glass placed at zero on the scale. A drop of blood is withdrawn and carried by means of a special capillary tube to the compartment opposite the unobstructed opening. The glass is then slowly moved along until the coloration of both compartments is the same. The percentage may then be read from the scale. In the average person, a registration of 85 to 90 percent on this scale may be considered normal. A darkened room, using a candle for illumination, is necessary for the best results by this method. The specific gravity method is more easily performed. Benzol and chloroform are mixed together forming a solution having a specific gravity of 1059. A drop of blood is placed in the mixture. Chloroform is added if it sinks, and benzol if it rises to the top, until the drop of blood is stationary, showing that it and the liquid are of the same density. The specific gravity is then taken in the usual way and the percentage of hemoglobin may be calculated from the following table by Hammerschlag:

Specific Gravity	=	Hemoglobin.
1033—1035	=	25—30 per cent.
1035—1038	=	30—35 ”
1038—1040	=	35—40 ”
1040—1045	=	40—45 ”
1045—1048	=	45—55 ”
1048—1050	=	55—65 ”
1050—1053	=	65—70 ”
1053—1055	=	70—75 ”
1055—1057	=	75—85 ”
1057—1060	=	85—95 ”

Hemoglobin is decreased in chlorosis and all forms of anemia and is said to be increased in pulmonary stenosis.

The number of blood cells is best determined by means of the Thoma-Zeiss hemocytometer. This apparatus consists of a glass slide with a central cell, the depth of which is 1-10 mm. Microscopic lines divide the floor of this cell into 400 squares each having a cubic capacity of 1-4000 mm. The surface of each square is 1-400 square mm. Double lines are used to mark off groups of 16 squares. Two pipets are used for diluting the blood, the one graduated to 100 is employed for the red cells while the one with the smaller scale is used for the white cells. Each pipet is blown into a bulb near one extremity to permit mixing of the blood with the diluting fluid. The diluting fluids commonly employed are normal salt solution, a two and a half percent of bichromate of potash solution, and Toison's fluid (methyl violet, 0.025 Gm., sodium chlorid, 1 Gm., sodium sulphate, 8 Gm., glycerin, 30 Cc., distilled water, 160 Cc.). A one-half percent solution of acetic acid is used in counting the white cells. The blood is diluted in the proportion of 1 to 100 or 1 to 200 in the enumeration of the red blood cells and in the proportion of 1 to 10 or 1 to 20 in determining the number of white blood cells.

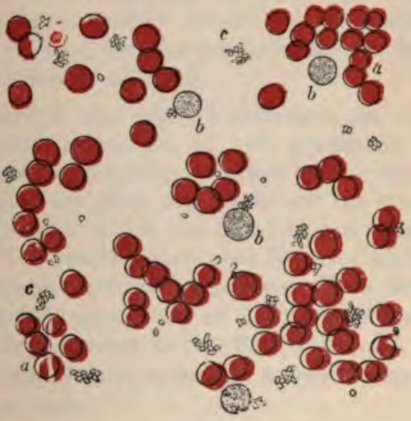

Fig. 23.—Blood Corpuscles, and Blood Plates, from Normal Human Blood.
a. Red Blood Corpuscles. *b.* Colorless Corpuscles. *c.* Blood Plates.
—(*von Jaksch.*)

In the practical application of this apparatus, the puncture is made in the skin and the drop of blood sucked up to the mark 1 Cc. on the pipet. The diluting fluid is then drawn into the tube until the mark 101 is reached (for red blood cells). The blood and diluting fluid are then carefully mixed and a drop of the mixture placed in the cell of the slide and covered by a cover-glass. After five or ten minutes the slide is placed under a microscope in a darkened room and the corpuscles counted by the aid of artificial light. In the calculation, the number of corpuscles counted in all the squares should be multiplied by 4000 and the product by the dilution. This entire product should then be divided by the number of squares counted, the quotient being the number of corpuscles in one cubic millimeter of blood.

The hematokrit is also used to determine the number of blood cells. It consists essentially of two graduated capillary tubes each 50 mm. long and having a caliber of 0.5 mm. mounted in a frame. The tubes are filled with blood and the apparatus attached to a centrifuge after which the blood is centrifugated for about two minutes. The result is then read from the scale, each division of which represents 100,000 corpuscles. This method is not to be used exclusively on account of its being inapplicable to white blood cells if their number is less than 20,000.

The white blood cells appear in several forms, each of which requires special study.

The small lymphocyte varies from 5 to 10 microns in diameter being nearly the same size as the red blood corpuscles. It is surrounded by a thin, scarcely visible ring of protoplasm. The nucleus is round and large and appears greenish-blue when stained by the acid fuchsin of Ehrlich's triple stain. In health these cells constitute from 20 to 30 percent of all the colorless blood corpuscles.

The large lymphocyte is a large mononuclear cell possessing the same characteristics as the preceding with the exceptions that the nucleus is round or oval and stains less deeply and the non-granular protoplasm is relatively larger in amount. The diameter of this cell may be as high as 13 to 15 microns. Transitional forms between the small and the large lymphocyte are also encountered. The percentage of large lymphocytes varies from four to eight.

The transitional forms resemble the foregoing except that their nuclei are indented, or horse-shoe shaped and the protoplasm is neutrophilic.

Polynuclear leukocytes are smaller than the large lymphocytes and occur in three forms. The *polymorphonuclear neuthrophiles* are matured leukocytes and constitute from 62 to 70 percent of the white blood cells. The nucleus is decidedly irregular and stains a greenish-blue or green with Ehrlich's triple stain. The protoplasm and nucleus contain fine granules which stain only with neutral stains. With Erhlich's solution they appear violet or purple, while the intervening matrix has a pinkish color. The *eosinophiles* are smaller than the neutrophiles, but contain larger granules which have a great affinity for acid stains such as eosin and the acid fuchsin of Ehrlich's triple stain. Eosin stains these granules a brilliant pink while the acid fuchsin of Ehrlich's stain causes them to assume a copper-red color. There may be more than one nucleus which may be recognized by their blue color in the presence of either of the already-mentioned stains. Eosinophiles may be considered as hypermature leukoyctes and constitute from one-half to four percent of the white blood cells.

The *basophiles* or *mast cells* contain granules which are unstained by Ehrlich's solution but which stain in basic solutions of the anilin dyes such as methylene-blue. These cells constitute from one-fourth to one-half percent of the white blood cells. *Myelocytes* are large, non-ameboid, white blood cells resembling the large granular cells of the bone-marrow. The nucleus is single and stains pale with the Ehrlich stain. The protoplasmic granules are usually neutrophilic.

The red blood cells in certain abnormal conditions undergo changes in size, shape, and characteristics. *Nucleated red blood corpuscles* are occasionally encountered and may appear as *normoblasts, megaloblasts,* and *microblasts.* Normoblasts are about the size of the ordinary red cells which they represent in the immature state. With the Ehrlich-Biondi stain the nucleus assumes a very deep blue color. Megaloblasts are large and irregular cells possessing large nuclei which stain pale green with the Ehrlich-Biondi solution. Microblasts appear as very small nucleated red blood cells.

Hemoconien or Müller's blood-dust, consists of small, round, highly refractive, colorless granules possessing molecular movements and resembling fat droplets.

Microscopic examination of the blood requires special preparation and staining of the specimen to obtain the best results. After the drop of blood is withdrawn it should be placed between two perfectly clean cover-glasses over each of which it then forms a thin film. After drying, the specimen is fixed by heat (100° to 110° C.) in a copper-box or blood-oven for a half-hour or more or by being placed in a mixture of equal parts of absolute alcohol and ether for about fifteen minutes. It may then be conveniently stained by immersion for a few minutes in a diluted one percent alcoholic solution of eosin. The excess of the stain is removed by washing the cover-slip in water after which it is counterstained with Delafield's hematoxylin solution for one minute. The specimen is again washed in water, dried, and mounted.

Ehrlich's triple stain may be employed instead. It is made up as follows: Saturated aqueous solution of orange G. 40 Cc., saturated aqueous solution of acid fuchsin, 45 Cc., saturated aqueous solution of methyl-green, 55 Cc., these are mixed together and to this mixture added distilled water, 50 Cc., alcohol, 50 Cc., glycerin, 15 Cc. The entire mixture should be kept in a cool, dark place for a week before being used. When stained with this solution the red cells assume an orange tint, the nuclei of the white cells appear greenish-blue, the neutrophilic granules are colored violet, and the eosinophilic granules are red.

ABNORMAL STATES OF THE BLOOD.

Oligocythemia is the term applied to diminution in the number of red blood cells irrespective of the cause.

Oligochromemia consists in a deficiency in the hemoglobin. It is usually proportionate to the reduction in the red blood cells except in chlorosis, in which the hemoglobin equivalent of each cell is greatly reduced, and pernicious anemia in which it is relatively high.

Leukocytosis is an increase of in the number of white blood cells with an excess of the polynuclear forms. It may be encountered as a physiologic process in pregnancy and parturition, in the new-born, during digestion, and after physical exertion. It is observed as a pathologic condition in leukemia, pernicious anemia, chlorosis, diseases of the lymphatic glands, inflammatory conditions associated with exudation, many of the infectious fevers, in malignant disease, in gout, uremia, and similar affections, after hemorrhage, and just before death. Drugs such as pilocarpin, ergotin, salicylates, and antipyrin, and also tuberculin induce leukocytosis. It is not present in uncomplicated cases of influenza, typhoid fever, typhus fever, malaria, measles, miliary tuberculosis, or tuberculosis unassociated with cavity-formation or enlargement of the lymphatic glands.

Eosinophilia is the term applied to any increase in the cells containing eosinophilic granules. It is observed in filariasis, trichinosis, ankylostomiasis, osteomalacia, and certain skin diseases.

Leukopenia is employed to designate a marked reduction in the number of white blood cells. It is observed in pernicious anemia.

Poikilocytosis is a condition characterized by irregularities in the shape of the red blood cells. They may be oval, pointed, angular, or reniform. It is seen in pernicious anemia and leukocythemia.

Microcythemia or microcytosis is the term applied to the condition in which the red blood cells are markedly reduced in size. It accompanies the severe anemias and toxemias.

Macrocythemia or macrocytosis is the opposite condition, the size of the red blood cells being greater than normal. It is associated with the severe forms of anemia especially pernicious anemia.

Nucleated red blood cells are abnormal constituents of the blood and are present in the grave forms of anemia. Their varieties and characteristics have already been described.

Hydremia is an excess of the watery constituents of the blood, with a corresponding decrease of the cellular elements. It is present in anemia, in anasarca, and after the ingestion of fluids in large quantities.

Anhydremia is a condition in which the fluid portion of the blood is greatly diminished. It occurs after excessive drains on the system from any cause as in hemorrhage and cholera.

Melanemia is a rare condition characterized by the presence of black, brown, or yellow granules in the blood. It is observed in malaria, relapsing fever, melanosarcoma, and Addison's disease.

Lipemia is applied to the presence of fat in the blood. It may be detected by the microscope as minute fat globules or by its black coloration when stained with a one percent solution of osmic acid. Lipemia occurs in chronic alcoholism, chronic nephritis, diabetes, pulmonary tuberculosis, and after injuries to the bone-marrow.

Parasites are encountered in the blood in certain diseases. They may be animal or vegetable parasites. The principal animal parasites are filaria sanguinis hominis, plasmodium of malaria, and distoma hematobium. The most important vegetable parasites are tubercle bacillus, streptococcus, staphylococcus, spirillum of relapsing fever, anthrax bacillus, typhoid bacillus, bacillus of glanders, colon bacillus, and the tetanus bacillus.

ANEMIA.

Definition.—A diminution in the number of red blood corpuscles or the entire quantity of the blood with alterations in its more important constituents such as albumin and hemoglobin. It may be local (ischemia) or general (oligemia), or it may be primary or secondary.

Causes.—The predisposing causes are female sex, pregnancy, menopause, heredity, and concealed foci of tuberculosis. The exciting causes are deficient food, air, or sunshine, excessive work, mental shock and anxiety, prolonged and frequent nocturnal emissions, excessive nursing, imperfect nutrition, chronic intestinal catarrh, prolonged discharges, hemorrhage, Bright's disease, parasites, malaria, syphilis, cancer, and various toxemias.

Pathologic Anatomy.—The blood is lighter in color, due to the reduction in the red cells and hemoglobin. It is thinner than normal and coagulates slowly and imperfectly on account of the diminution in the fibrino-plastic constituent. After death the tissues are thin, shrunken, and bloodless and if the anemia has been of long duration patches of fatty degeneration will be observed in the various organs.

Symptoms.—Pallor of the skin and various mucous membranes is marked. Muscular weakness and loss of strength are present. Febrile paroxysms are not uncommon. The appetite is impaired and there is imperfect digestion with occasional attacks of vomiting. Respiration is quickened. There are also irritability of temper, vertigo, swooning, hysteria, and epileptoid attacks. The pulse is rapid and full, and the heart is irritable with systolic basic murmurs. The cervical vessels pulsate and there is a hum over the jugular vein. There may be extravasations of blood into the mucous membranes. Nocturnal emissions in the male and deficient menses in the female accompany the condition. In children marasmus is common. Edema of the ankles is often present. In long-continued cases, symptoms of fatty change in the various organs or gastric ulcer may appear. Examination of the blood reveals a reduction in the number of red cells with changes in size and shape, nucleated blood cells, diminuton in hemoglobin, and an increase in the number of white cells.

Prognosis.—In secondary anemias, those in which the cause can be ascertained and promptly overcome the outlook is favorable in the absence of complications and degenerative changes. In the primary anemias, such as chlorosis, pernicious anemia, leukemia, Hodgkin's disease, and splenic anemia, the prognosis is less favorable. These forms will be fully described later.

Treatment.—The cause should be removed and rest, restricted exercise, fresh air, sunlight, and a highly nutritious diet should be advised. The various symptoms should be met with suitable remedies as they arise. The tonics of most value in this condition are iron, arsenic, quinin, and strychnin. The carbonate of iron, gr. ii-v (0.13-0.32 Gm.), is most often employed but Blaud's pill, Basham's mixture, tincture of the chlorid of iron, or other preparations of iron may be used. Great care should be exercised to prevent constipation while administering any of the iron preparations.

The following alternative tonic, known as Smith's (Dr. A. H.) "four chlorids," is frequently of value:

℞. Hydrargyri chloridi corrosivi	gr. j-ij	.065-.13	Gm.
Liq. arsenici chloridi	fʒ j	4.	Cc.
Tinct. ferri chloridi Acidi hydrochlorici dil.	āā fʒ iv	āā 15.	Cc.
Syrupi	fʒ iv	15.	Cc.
Aquæ	ad f℥ vj	180.	Cc.

M. S.—One dessertspoonful in a wineglass of water after each meal.

Cases of anemia with weak stomach can take the following "iron lemonade" without discomfort:

℞. Tinct. ferri chloridi ...fʒ ij	8. Cc.
Acid phosphor. dil. .....fʒ ij	8. Cc.
Syr. limonis ....f℥ ss	15. Cc.
Aquæ ....f℥ ij	60. Cc.

M. S.—One teaspoonful, well-diluted, after meals.

CHLOROSIS.

Synonyms.—Essential anemia; green sickness.

Definition.—A pronounced anemia met with chiefly in young girls about the age of puberty, characterized by diminution in the percentage of hemoglobin.

Causes.—The exciting cause is unknown. Puberty, female sex, overwork, impure air, improper food, lack of sunshine, prolonged lactation, menstrual disorders, heredity, emotional disturbances, change of climate, and constipation are important predisposing factors.

Pathologic Anatomy.—The number of red blood corpuscles is nearly normal but there is marked decrease in the hemoglobin sometimes as low as twenty percent. The body is usually well-nourished and the subcutaneous fat well-distributed. There is pallor of the organs and muscles but there are no other alterations in the spleen, lymphatic glands, or bone-marrow. The circulatory apparatus is usually imperfectly developed, the heart and arteries being congenitally small. The genitalia are often immature.

Symptoms.—Frequently the attention is first called to the condition at the time of some menstrual disturbance such as amenorrhea or menorrhagia. Coincidently or shortly after such an attack the complexion changes, blondes becoming pallid, waxy, and puffy without edema, while brunettes assume a muddy or grayish color with bluish black rings under the eyes. Weakness and fatigue manifest themselves on the slightest exertion. Shortness of breath is common. The heart is irritable and the pulse soft and full. The peripheral veins may pulsate. The patient experiences a change of disposition becoming morose and despondent, hysterical, or melancholic. The appetite is capricious and perverted and digestion is impaired. Attacks of gastralgia are frequent and gastric ulcer or phthisis may occur as complications. There is no loss of flesh; the patient on the contrary appears somewhat stout. The yellowish-green tinge of the skin is characteristic. Functional cardiac murmurs may be detected and a hum may be heard over the jugular vein especially the right. Febrile attacks are not infrequent. Headache and neuralgia may also be present. Constipation is a common accompaniment.

Examination of the blood shows a relative decrease in quality and quantity of the hemoglobin, resulting in the blood being paler than normal. The red corpuscles are also lighter in color and show less tendency to form rouleaux; their character also changes, not all being of uniform size, some normal, others small (microcytes), others unusually large (macrocytes), others irregularly shaped (poikilocytes). The number may be normal 5,000,000 to the cubic millimeter, or the number is occasionally increased, but it is usually lessened, there being as few as 3,000,000 or 2,000,000. The white corpuscles are usually normal in number, but in some instances their number is increased (leukocytosis). Rarely granular bodies are found in the blood which are generally regarded as the products of the degeneration of the white blood corpuscles.

Complications.--The principal complications are gastric ulcer, phthisis, menstrual disorders, gastroptosis, and venous thrombosis.

Diagnosis.—An examination of the blood usually renders the diagnosis very easy but if impracticable for any reason, the peculiar color in young girls associated with weakness and various functional disorders should lead one to suspect the presence of chlorosis and treat accordingly. Tuberculosis, peptic ulcer, lead-poisoning, nephritis, etc., may be distinguished from it by exclusion.

Prognosis.—Death is a very rare termination. The affection is chronic and shows a great tendency towards relapses and complications.

Treatment.—The unhygienic surroundings that usually attend these cases should receive attention. The quality of the food should be improved and the patient should receive an abundance of fresh air and sunshine, and regulated exercise should be advised. Work should be interdicted. Iron should be administered in some form being careful to guard against its constipating effect by the use of some laxative. The tincture of the chlorid of iron is usually employed.

J. W. England, Ph. G., has proposed the following formula for an iron pill that has been successfully used at the Philadelphia Hospital:

℞.	Massæ ferri carb.	gr. xlviij	3. Gm.
	Potassii sulph.	gr. xxxiv	2. Gm.
	Potassii carb.	gr. v⅓	.33 Gm.
	Pulv. altheæ	gr. iss	.02 Gm.
	Pulv. acaciæ	q. s.	q. s.

Ft. pil No. xvj, and inclose in gelatin capsules.
M. S.—One three times daily.

The following is Blaud's formula:

℞.	Pulv. ferri sulph.		
	Potassii carbonat. puræ	āā℥ ss	āā 15. Gm.
	Tragacanthæ	q. s.	

Ft. pil No. xcvj.
M. S.—One to three or four pills three times daily.

The addition of arsenic is necessary in some cases. The following formula is valuable under such circumstances:

℞.	Ferri arseniatis	gr. 1-12-⅙	.005-.011 Gm.
	Ext. nucis vomicæ	gr. ⅙-¼	.011-.016 Gm.

Ft. pil No. j.
M. S.—After meals.

Or—

℞.	Liq. arsenici chloridi ..	fʒ ij	8. Cc.
	Tinct. ferri chloridi	fʒ vij	28. Cc.
	Glycerini	f℥ j	30. Cc.
	Elix. aurantii ..q. s. ad	f℥ iij	90. Cc.

M. S.—One teaspoonful after meals, in water.

The saline laxatives and the alkaline mineral waters are useful adjuncts to the treatment. Dilute hydrochloric acid, manganese, and phosphorus may also be employed. The blood should be examined from time to time in order to note the effects of treatment.

PROGRESSIVE PERNICIOUS ANEMIA.

Synonyms.—Idiopathic anemia; anematosis; essential anemia; anemia of fatty heart.

Definition.—A progressive, pernicious form of anemia, of unknown cause, usually resisting all treatment, and toward its termination associated with fever.

Causes.—The exciting cause of the disease is unknown. It probably results from the action of some toxin generated in the digestive tract. Pregnancy, syphilis, intense mental anxiety and worry, middle life, and male sex seem to predispose towards this affection. Intestinal parasites (Bothriocephalus latus and Ankylostomum duodenale) are responsible for some cases.

Pathologic Anatomy.—The blood is scanty and pale, with alterations in the size, shape, and number of the red blood cells and diminished hemoglobin. It coagulates very slowly and imperfectly. There is no increase in the white corpuscles. The bone-marrow becomes fetal, red, and adenoid in character containing nucleated red blood cells, macroblasts, neutrophiles, and eosinophiles. There is a deposition of iron pigment in the liver cells and those of the spleen. The heart, larger arteries, liver, spleen, kidneys, stomach, and muscles exhibit circumscribed or diffused fatty degeneration. There is not much emaciation although pallor of the surface of the body is pronounced. The muscles are often unusually red. Changes in the ganglion cells of the sympathetic, sclerosis of the posterior columns of the cord, softening of the lumbar segment, and atrophy of the gastric mucous membrane may be encountered coincidently. Hemorrhages into the skin, mucous membranes, and retina may also be observed.

Symptoms.—The affection begins insidiously with increasing langour and pallor, the muscular weakness compelling the patient to take his bed. Cardiac palpitation, dyspnea, attacks of syncope, edema, and swelling of the ankles follow. Petechial hemorrhages scattered irregularly over the body-surface make their appearance and there is often tenderness over the sternum and other superficial bones. The weakness progresses but emaciation is absent. The pulse is large, soft, and jerky; nervous pulsations are observed; and hemic murmurs are often heard. There is loss of appetite, and nausea, vomiting, and diarrhea may occur. Disorders of vision are not uncommon and are due to retinal hemorrhages. As the disease progresses, a remittent form of fever develops, the temperature frequently rising to 102°-104° F.

Blood Changes.—The number of red blood cells is decidedly reduced, sometimes it is as low as 500,000 per cubic millimeter, but the hemoglobin is not correspondingly diminished although its entire quantity is considerably less than normal. The red cells show marked alterations in size, shape, and characteristics. They may be large and ovoid (megalocytes) or they may be small, round, and of a deep red color (microcytes). Some of the red corpuscles are markedly irregular and to these, the term "poikilocytes" is applied. Nucleated red cells (normoblasts and megaloblasts) are almost constantly present. The blood plaques are scanty or absent. The leukocytes are usually normal or slightly diminished with a relative increase in the small mononuclear forms. Eichhorst's corpuscles may be encountered.

Diagnosis.—The microscopic examination of the blood, showing the marked variations in size, shape, and characteristics and the enormous reduction in the number of the red cells serves to make the diagnosis in all cases, as the symptoms while suggestive are by no means conclusive since they are more or less common to other forms of primary anemia.

Prognosis.—The disease terminates in death usually within one or two years after its recognition. Remissions are common. Recovery may occur in parasitic forms of the disease on removal of the cause.

Treatment.—The treatment is unsatisfactory. Rest in bed, fresh air, good food, salt baths, massage, and similar hygenic measures should be prescribed. Arsenic is of value as it seems to check the progress of the affection. It should be administered to the point of tolerance. Iron should also be given alone or combined with arsenic. Inhalations of oxygen are recommended and bone-marrow is sometimes employed internally. When due to intestinal parasites, anthelmintics should be administered.

LEUKOCYTHEMIA.

Synonyms.—Leukemia; white cell blood; white blood; anemia splenica.

Definition.—A condition in which there is an enormous increase in the number of white blood corpuscles, with enlargement of the lymphatic glands, spleen, and often of the bone marrow—viz.: *splenic, lymphatic,* or *myelogenic,* and is characterized by symptoms of pronounced anemia.

Causes.—The true cause of the disease is unknown. It occurs with greatest frequency in middle-aged males and is believed to be influenced by heredity, traumatism, syphilis, and pregnancy. By some observers it is considered to be of infectious origin.

Pathologic Anatomy.—The morbid anatomy of leukemia includes alterations in the blood, the spleen, the lymphatic glands, and the bone-marrow. According to the predominance of the organic lesions, it is termed splenic, lymphatic, or myelogenic. Most cases, however, are combinations of these varieties.

The spleen is increased in size, density, and firmness and shows hyperplasia of its lymphoid structure. The organ may be adherent to neighboring structures and is often the seat of lymphoid tumors. The liver is also enlarged and infiltrated with leukocytes. The lymphatic glands all over the body enlarge but are soft to the touch, often fluctuating, and movable. The solitary glands, Peyer's patches, the tonsils, and the lymph-follicles of the tongue, pharynx, and mouth may partake of similar alterations. The red bone-marrow reverts to the embryonal type. Its color becomes greenish-yellow and the fat disappears. It often becomes pyoid in appearance. The microscope will reveal large numbers of nucleated red blood cells in varying stages of development, polynuclear and mononuclear leukocytes, eosinophiles, and large neutrophiles with single nuclei (myelocytes). The blood is paler than normal; its specific gravity is reduced to 1040 or lower; the white cells are increased in number and size; and the red corpuscles are reduced in number and size.

Symptoms.—The onset is insidious and in the early stages the manifestations are identical with those of simple anemia. There is also swelling of the abdomen and a feeling of fullness and pain in the region of the spleen due to enlargement of that organ. In

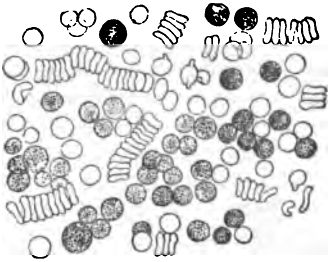

FIG. 24.—LEUKEMIC BLOOD. (*Landois.*)

the lymphatic variety there is enlargement of the glands with pallor of the body-surface. In the myelogenic variety, the bones especially the ribs and sternum are tender on pressure and the patient assumes a waxy appearance. The liver and spleen are enlarged and moderate fever may be present. In all varieties, emaciation, weakness, loss of appetite, feeble digestion, diarrhea, cardiac palpitation, dyspnea, and edema of the ankles and eyelids are observed. Hemorrhages into the skin or from the mucous membranes may be early symptoms or may occur near the termination of the disease. Priapism is an occasional manifestation of this disease. The urine is high-colored, scanty, of high specific gravity (1020-1030), and often slightly albuminous.

The Blood.—The blood is pale and watery and the white blood corpuscles are enormously increased in number. The leukocytes in some cases equal the red blood cells in number and in rare cases exceed them. There is also a reduction in the entire number of blod cells. The blood coagulates slowly, and its specific gravity and alkalinity are subnormal. The addition of a drop of dilute gentian-violet solution stains the white cells blue and they may then be readily detected. Nucleated red cells and poikilocytes are present and the hemoglobin is diminished. Blood plaques are somewhat increased. Charcot's crystals are often present. In *Splenic-myelogenous leukemia* the small mononuclear leukocytes are relatively diminished. The proportion of eosinophiles is unchanged. The polynuclear neutrophiles undergo marked alterations in size but their proportion usually remains normal. The characteristics of this form are the cells derived from the bone-marrow (myelocytes) containing neutrophilic granules, and large mononuclear cells containing karyokinetic figures. Polynuclear basophilic cells are constantly present in great numbers. Nucleated red cells, (gigantoblasts) and cells with fragmented nuclei may be encountered. *Lymphatic leukemia* is rare and may be acute or chronic in form. The acute form occurs usually in children and is attended by lymphatic enlargement, hemorrhages, and the presence of large pale lymphocytes in excess in the blood. The chronic form is characterized by an increase of the white blood cells but to a less extent than the splenic variety of the disease. The small lymphocytes are affected especially by this increase, the other forms of leukocytes being relatively diminished. Eosinophiles, myelocytes, and nucleated red cells are rarely present.

Diagnosis.—As in other affections associated with symptoms of anemia, microscopic examination of the blood is absolutely necessary to determine the true nature of the disease. The enormous increase in the white cells with changes in their size, characteristics, and proportion, and the presence of new cellular elements in the blood are the distinctive features. Leukocytosis is attended primarily by an increase of the white cells but this increase is not so great as in leukemia and affects chiefly the polymorpho-multinuclear neutrophiles. The variations in the characteristics of the cells will aid in distinguishing the varieties of leukemia one from the other.

Prognosis.—Acute leukemia terminates fatally within two or three months; the other forms seldom last more than two or three years and also end in death.

Treatment.—While seldom satisfactory, the various symptoms should be treated as they arise on general therapeutic principles. Rest, nutritious diet, fresh air, sunshine, and cod-liver oil, hypophosphites, iron, quinin, strychnin, arsenic, ergot, or oxygen should be prescribed. X Ray is best treatment. Often cures {Sen

HODGKIN'S DISEASE.

Synonyms.—Pseudo-leukemia; Pseudo-leukocythemia; lymphatic anemia; lymphadenoma.

Definition.—An affection characterized by hypertrophy of the lymphatic glands in various parts of the body, associated with marked anemia.

Cause.—Unknown.

Pathologic Anatomy.—A hyperplasia of the lymph glands interfering more or less with their functions. The enlargement may be confined to one isolated gland, or a number may be affected in different portions of the body, or a number in one location may be simultaneously affected, causing a tumor varying in size from an egg to an orange or even larger. The spleen and liver are involved in two-thirds of the cases. "The marrow of the long bones may be converted into a rich lymphoid tissue." (Osler.) The red blood corpuscles are decreased in number and altered in size and shape; the white blood corpuscles are often increased in number.

Symptoms.—A slowly developing anemia with isolated or diffused enlargement of the lymphatic glands. As the condition develops, fever of a remittent character occurs, with feeble cardiac action and shortness of breath. Hemorrhages may occur. The patient grows progressively worse with all the associated symptoms of deficient blood, death occurring by asthenia.

Diagnosis.—A study of the clinical history will prevent error, as tubercular or scrofulous glands are accompanied with tubercular changes elsewhere, and do not present the same blood-changes as Hodgkin's disease.

Prognosis.—Unfavorable. The progress may be slow, but it is none the less toward a fatal termination. The duration is from two to three years.

Treatment.—The treatment is that of pernicious anemia. Surgical intervention is sometimes necessary to relieve the dyspnea. Radiotherapy may be of value in some cases.

STATUS LYMPHATICUS.

A rare disease of the blood-making organs occurring in children and young persons, characterized by a hyperplasia of the lymphoid tissues throughout the body, including the lymphatic glands, spleen, thymus gland, and bone-marrow. The symptoms are secondary to the nutritional disturbances. The affection resembles Hodgkin's disease very closely.

SPLENIC ANEMIA.

An anemic condition in which the spleen is greatly enlarged and indurated and its lymphatic structure is destroyed and replaced by an overgrowth of the reticulum. The red blood cells and hemoglobin are diminished. Microcytes, megalocytes, poikilocytes, and numerous nucleated red blood corpuscles are present. The leukocytes are slightly increased. The disease lasts from six months to three years and terminates in death. The treatment is unsatisfactory. Removal of the spleen has been followed by recovery.

ADDISON'S DISEASE.

Synonym.—Melasma suprarenalis.

Definition.—"The bronzed-skin disease." Thus defined by Averbeck: "A well-marked constitutional disease, exhibiting itself locally as chronic inflammation of the suprarenal capsules, but in its essence consisting in a peculiar anemic condition, always tending toward death, and characterized by intense development of pigment in the cells of the rete malpighii and in the epithelium of the mucous membrane of the mouth, and extreme debility."

Causes.—Obscure. Tubercule, scrofula, and syphilis have each been given as the cause. It is usually encountered in middle-aged men.

Pathologic Anatomy.—A low form of inflammation, terminating in degeneration of the suprarenal capsule. In the majority of cases the affection is tuberculous in character although carcinomatous changes are also observed at times. The blood is deficient in fibrin and red corpuscles, with a slight increase of the white corpuscles. Fatty degeneration of the heart and vessels has been observed in some cases.

"The most striking change during life—the abnormal pigmentation—is due to the deposition of granular pigment in the cells of the rete malpighii, in the papillary portion of the cutis, and even in the connective-tissue corpuscles. No change occurs in the proper structure of the skin. Similar pigment deposits occur in the mucous membrane of the mouth, especially along the edges of the teeth."

" The disease of the suprarenal capsules excites an irritation of the vasomotor system—the trophic system—which leads to the pigmentation."

Symptoms.—The onset of the disease is insidious, with a feeling of extreme languor, muscular fatigue, asthenia, indigestion, anorexia, dyspnea, cardiac palpitation, vertigo, melancholia, and excessive drowsiness. The surface is first pale, then changes to a hue like that of melanemia, changing to icteroid, finally resembling the color of a mulatto, and then to a lusterless bronze. These changes also occur on the mucous membrane of the lips, tongue, gums and mouth.

Prognosis.—An incurable disease. Duration, a year or two.

Treatment.—Symptomatic. The administration of suprarenal extract, gr. iii-v, three times daily has been followed by temporary improvement.

HEMOPHILIA.

Synonyms.—Hemorrhagic diathesis; "bleeder's disease."

Definition.—A congenital condition characterized by a tendency to uncontrollable hemorrhages, with or without abrasions.

Cause.—Heredity is the most prominent etiologic factor. Males are most often affected, the disease being transmitted by females.

Symptoms.—The bleeding appears about the period of first dentition, and consists of spontaneous hemorrhages from the mucous membrane of the nose, mouth, lungs, stomach, intestines, and genitourinary passages, or in typical cases hemorrhages occur directly from the fingers, toes, lobes of the ears, back of the hands or arms, without any apparent change in the skin, and continue in spite of the most powerful means, for days or weeks. Traumatic hemorrhages occur if an injury of any kind is sustained about the period of the development of the bleeding. Epistaxis is common. Attacks of arthritis with fever frequently occur.

Prognosis.—The condition usually terminates fatally before puberty. Death rarely occurs in the first bleeding. The younger the individual at the time of the development of the disease the more unfavorable is the prognosis. The hemorrhagic tendency may be outgrown.

Treatment. — Internally, the administration of potassium chlorate, tincture of the chlorid of iron, and ergot are believed to favorably influence the condition. Locally, pressure, ice, heat, gelatin, tannic acid, gallic acid, calcium chlorid, fibrin ferment, adrenalin, etc., may be employed.

SCORBUTUS.

Synonyms.—Scurvy; scorbutic purpura.

Definition.—A peculiar condition of malnutrition or anemia, characterized by great debility, mental lethargy, hemorrhages of the skin and from the mucous membranes, and a swollen and spongy condition of the gums which tend to bleed on the slightest irritation.

Causes.—A deficiency of fresh vegetables in the diet, and unhygenic surroundings are the most common factors in its production. Mental depression, home-sickness (nostalgia), and similar disturbances also seem to influence the condition. It occurs most often in sailors of the merchant marine, in prisons, in armies, and where large bodies of men are collected under unsanitary conditions. By some observers the disease is considered infectious.

Pathologic Anatomy.—The blood is dark and fluid, and its composition deranged with diminution in the potassium salts. Anemia is present but there is no increase in the white blood cells. The structure of the blood-vessels is altered allowing spontaneous hemorrhages into the skin, muscles, joints, and internal organs and from the mucous membranes. There is swelling and ulceration of the gums often resulting in loss of the teeth. Ulcers of the ileum and colon may be encountered. The spleen is enlarged and soft and the viscera are affected with parenchymatous changes.

Symptoms.—The onset is slow, with general weakness, lassitude, indisposition to mental or physical exertion, and anemia. The skin is rough, dry, and of a muddy pallor and the face is pale and bloated. The gums soon become swollen and spongy and may present a fungoid appearance. They tend to bleed on the slightest irritation. The teeth loosen and may fall out. The breath is extremely offensive. The lips are pale and the eyes are sunken and surrounded by dark blue circles. There is marked depression of spirits. Palpitation of the heart and dyspnea follow slight exertion. Petechial hemorrhages of the skin and bleeding from the various mucous surfaces are common. Brawny induration of the muscles is often observed. Edema of the face and ankles is not infrequent. Pain, tenderness, and swelling of the joints may be present. Various visual disturbances may occur. Fever is absent except in the late stages and in the presence of complications. Constipation is common. The urine is high-colored, of increased density, and contains an increase of phosphates and often blood.

Infantile Scurvy (Barlow's disease) occurs in young children as the result of improper feeding, usually following the long-continued use of proprietary foods, condensed milk, etc. Prostration, anemia, and general debility are marked. In the beginning the child lies with the legs drawn up and immobile, any attempt to move them inducing pain and consequent crying. They are not tender at this stage but soon ill-defined swellings appear on the lower extremities and extreme tenderness is manifest. The limbs are now everted and immobile (pseudoparalysis). Weakness becomes profound and hemorrhagic extravasations may be noted. Epiphyseal fractures are common. The sternum and adjacent cartilages appear depressed and localized thickening of the various bones of the body may be observed. Proptosis of one eyeball, puffiness of the upper lid, and subconjunctival ecchymoses are not infrequent.

Complications.—Dysentery, typhoid fever, or typhus fever may exist coincidentally.

Prognosis.—Recovery is the rule, both in adults and infants, when the appropriate treatment is instituted early in the course of the disease.

Treatment.—The patient should be removed to sanitary quarters and fresh vegetables added to the diet. The principal antiscorbutic substances are raw cabbage, cresses, potatoes, sauerkraut, onions, lemon-juice, oranges, and various fruits. These should be employed in conjunction with meats, milk, and farinaceous foods. In the infantile form the artificial feedings should be properly adjusted by the attending physician and in addition orange-juice should be administered. In all forms the mouth should be thoroughly cleansed with mild antiseptic and astringent lotions. Iron, quinin, strychnin, and the bitter tonics may be necessary to stimulate the appetite and combat the exhaustion.

PURPURA.

Synonyms.—Hemorrhea petechialis; Morbus maculosus Werlhofii.

Definition.—An acute disease, characterized by purplish discolorations of the skin, the result of hemorrhages into the upper layers of the cutis and beneath the epidermis. When the purpuric spots are tiny, like a pin-point, they are termed petechiæ; when larger in size, they are termed ecchymoses.

Varieties.—*Purpura simplex; purpura hemorrhagica; purpura urticans; peliosis rheumatica.*

Causes.—The etiology is obscure. The disease occurs most frequently in debilitated individuals. In many cases it is secondary to some other affection, but it also occurs as a primary condition. By some observers it is believed to be of infectious origin.

Symptoms.—*Purpura simplex* is the mildest form of the affection, and is characterized by the sudden appearance of small, bright red spots—a cutaneous hemorrhage—most commonly on the legs, coming in crops, associated with slight lassitude, mild febrile reaction, and aching pains in the limbs. The hue of the spots rapidly fades to a purplish color and they slowly disappear. Relapses are common.

Purpura hemorrhagica has, in addition to the eruption of purpura simplex,—the cutaneous hemorrhage,—a flow of blood from the free surface of mucous membranes. The most common hemorrhage is epistaxis, slight or profuse. Other hemorrhages are hematemesis, melena, hematuria, hemoptysis, menorrhagia, and also into the substance of the mucous membranes of the palate, cheek, and gums. This variety is associated with great debility and depression, moderate fever, and disorders of digestion. Marked anemia results from the hemorrhages.

Purpura urticans is a combination of urticaria and purpura simplex. It is characterized by rounded and reddish elevations of the cuticle, resembling wheals, but which are not accompanied, like the wheals of urticaria, by any sensation of itching or tingling. They are usually situated on the legs, thighs, breast, and arms, and are interspersed with petechiæ. They gradually form and subside within twenty-four or thirty-six hours. Relapses are frequent. This variety is also associated with malaise, moderate fever, and pains in the limbs.

Peliosis rheumatica (Schönlein's disease) is characterized by multiple arthritis and a purpuric eruption; frequently the arthritic symptoms are associated with urticaria or with erythema exudativum. Edema is often marked, as are also the fever, sore-throat, and general constitutional symptoms. The eruption is sometimes of vesicles—pemphigoid purpura.

Diagnosis.—Purpuric spots may be distinguished from other lesions of the skin by their failure to entirely disappear on pressure. The concomitant symptoms will serve to separate the several varieties and to distinguish the disease from scurvy and hemophilia.

Prognosis.—Purpura simplex and purpura urticans are favorable, but relapses are very frequent. Purpura hemorrhagica is always a grave disease, often proving fatal from exhaustion, or, more rarely, from cerebral or pulmonary hemorrhage. Peliosis rheumatica is often a severe affection, but recovery is the rule.

Treatment.—Rest in bed with the administration of a concentrated nutritious diet, tonics, and stimulants is necessary when there is much depression. Mild cases, rheumatic in origin, do well with the use of the salicylates and potassium iodid. In marked cases, arsenic, dilute sulphuric acid, tincture of the chlorid of iron, ergot, silver, nitrate, digitalis, quinin, turpentine, and similar remedies will be required. The following formula may be employed:

℞.	Ol terebinthinæ ....fʒ ij	8. Cc.
	Ol. amygdalæ express. ..f℥ j	30. Cc.
	Tinct. opii deodorat. ...fʒ ss	2. Cc.
	Mucil acaciæ ....f℥ j	30. Cc.
	Aq. lauro-cerasi.....ad f℥ iij	ad 90. Cc.

M. S.—One tablespoonful every three hours, diluted.

GENERAL DISEASES.

ACUTE ARTICULAR RHEUMATISM.

Synonyms.—Rheumatic fever; inflammatory rheumatism.

Definition.—A constitutional disease, characterized by fever, inflammation in and around the joints, acid sweats, and a great tendency to inflammation of either the endocardium or pericardium.

Causes.—The disease is believed by many observers to be of infectious origin but no specific organism has been as yet isolated. Certain predisposing factors seem necessary for the production of the disease. Among these may be mentioned exposure to cold and wet, sudden reductions in the temperature, lowered vitality from various causes, winter and spring seasons, heredity, infectious fevers, especially scarlet fever, puerperium, male sex, and previous attacks. The affection is seldom observed before seven or after fifty years of age.

Pathologic Anatomy.—The affected joints are intensely congested and the synovial membrane and surrounding ligamentous tissues are greatly swollen. The cartilage may be eroded. The synovial fluid is thinner than normal and of a reddish color, containing albumin, some gelatinous coagula of fibrin, leukocytes, but no pus cells or organisms in simple cases. There is an increase in the quantity and in the number of white corpuscles in the blood. The inflammatory edema of the joint and adjacent structures gives rise to considerable visible swelling and by its stretching of the parts and pressure on the nerves induces, in all probability, the pain. The joint condition usually ends in resolution. The complications of this affection possess no features different from the same conditions when occurring independently.

Symptoms.—Usually the onset is abrupt, generally at night, with a chill or chilliness, pain and stiffness in the joints, loss of appetite, and at times nausea and vomiting, followed by fever, the temperature soon reaching 102° to 104° F., and in rare cases 108° to 110° F. In some cases it is preceded by slight malaise, vague pains in the joints, and tonsillitis. After the affection has begun, there are profuse acid sweats, great thirst, constipation, and scanty, high-colored, acid urine containing an excess of uric acid and urates, and sometimes traces of albumin. The fever continues throughout the attack, often with marked remissions. Delirium is absent except when hyperpyrexia is present. Sleep is prevented by the pain and the profuse perspiration. The strength is moderately well-preserved. The skin is covered with various forms of miliaria or prickly heat due to excessive irritation of the sweat-glands.

The local phenomena are pain, increased by motion and pressure, tenderness, and increased heat, swelling, and redness of one or more joints. Swelling is most apparent in those joints not covered by muscle as the knee, wrist, elbow, and the ankle and is proportionate to the acuteness of the attack. The inflammation may suddenly cease in one or more joints and abruptly appear in others.

The disease is extremely irregular as regards the number of joints affected, although the local manifestations are controlled by an important pathologic law, *the law of parallelism.* The affected joints are either on one side of the body, or those on both sides that are analogous, as the knee, wrist, ankle, hip, and shoulder, are attacked together. The migratory character of the inflammation is especially distinctive.

In some cases the affection is unattended by articular manifestations.

Complications.—The most common complications are endocarditis, pericarditis, myocarditis, cerebral endarteritis, pleurisy, peritonitis, bronchitis, pneumonia, tonsillitis, hyperpyrexia, erythema nodosum, urticaria, and purpura. As sequels may be mentioned chorea, acute nephritis, false ankylosis, chronic rheumatism, and exophthalmic goiter.

Duration.—The duration of acute rheumatism is governed entirely by the presence or absence of complications. Uncomplicated cases recover in from thirteen to twenty-one days, although they may be prolonged to five or six weeks. Relapses are frequent.

Diagnosis.—A typical case cannot be mistaken for any other disease, but cases running a subacute course may be mistaken for acute rheumatoid arthritis, gonorrheal rheumatism, or pyemia.

Acute rheumatoid arthritis attacks one joint at a time and becomes permanent, has slight, if any fever, and no sweats or cardiac lesions.

Gonorrheal rheumatism is associated with a gleety discharge, or follows the sudden cessation of an acute or subacute gonorrheal discharge, attacks either the ankle or wrist only, is slowly influenced by treatment, and lacks the febrile phenomena.

Pyemia is usually manifested in a single joint at the time, and is followed by suppuration and all the symptoms of hectic fever.

Prognosis.—Recovery is the rule in uncomplicated cases, the mortality being about three per cent. When death occurs, it usually depends upon hyperpyrexia, cardiac complication, or cerebral endarteritis.

Treatment.—In all cases the patient should be placed at absolute rest in bed. The patient should wear woolen garments and blankets (no sheets) should constitute the bed clothing, being careful to protect the inflamed joint from excessive weight of the coverings. The diet should consist of easily-digested substances preferably milk. The free use of water particularly the alkaline mineral waters should be encouraged. In strong and vigorous patients the administration of salicylic acid or the salicylates in large and frequently repeated doses is of great benefit. Sodium salicylate, ammonium salicylate, strontium salicylate, salicin, salol, salophen, or oil of wintergreen may be used pushed to the point of tolerance. In all cases, special internal treatment should be preceded by a course of calomel followed by a saline laxative.

℞. Acidi salicylici.........℥ ss	15. Gm.
Liq. ammonii acetat. ...f℥ iv	120. Cc.
Spts. ætheris nitrosi ...f℥ j	30. Cc.
Syr. simplicis ....f℥ j	30. Cc.

M. S.—Tablespoonful every three hours, well-diluted.

Or—

℞. Sodii salicylat. ....℥ j	30. Gm.
Tinct. cinchonæ comp...f℥ iij	90. Cc.
Aq. menth. pip. ......f℥ iij	90. Cc.

M. S.—Dessertspoonful every three or four hours till relief, when the interval should be increased.

Or—

℞. Potassii acetat. ....℥ j	30. Gm.
Acid salicylici ....℥ ss	15. Gm.
Syr. limonis ....f℥ ij	60. Cc.
Aq. menth. pip. ....f℥ viij	240. Cc.

M. S.—Tablespoonful every three hours, diluted.

Usually this treatment affords rather prompt relief but if after three or four days' trial there is no benefit derived from it, alkaline treatment should be substituted. This consists in the administration of an ounce and a half of one of the alkaline carbonates, either alone or combined with a vegetable acid, every twenty-four hours until the urine becomes neutral or alkaline when the quantity should be reduced to an amount just sufficient to maintain alkalinity. The following formulas are frequently employed:

℞. Potassii bicarbonatis ....ʒ ij	8. Gm.
Acid. tartarici ....gr. xxx	2. Gm.

M. S.—Dissolve in a glass of water and drink effervescing every three hours.

Or—

℞. Potass. bicarb. ....ʒ ij	8. Gm.
Succus limonis ....fʒ iv	15. Cc.
Aquæ chloroformi .....f℥ ss	15. Cc.

M. S.—In water, every three hours.

After the acute symptoms have subsided, Basham's mixture or tincture of the chlorid of iron, ♏ xx (1.3 Cc.) every three hours, should be administered. In pale, feeble, and anemic patients the following prescriptions will be of great value:

℞.	Strychninæ sulph.	gr. 1-60	.001 Gm.
	Tinct. ferri chlor.	♏ xv-xxx	1.-2. Cc.
	Liquor. ammonii acetat .	f℥ ss	15. Cc.

M. S.—Every four hours, in a glass of water.

Or—

℞.	Sodii salicylatis	ʒ iv	15. Gm.
	Glycerini	f℥ j	30. Cc.
	Acidi citrici	gr. x	.6 Gm.
	Ol. gaultheriæ	fʒ ss	2. Cc.
	Mucil. acaciæ	f℥ ss	15. Cc.
M.	Add while stirring		
	Tinct. ferri chlorid.	fʒ iv	15. Cc.
	Liq. ammonii citrat. (B. P.)	ad f℥ iv	ad 120. Cc.

M. S.—One to two teaspoonfuls every two, three, or four hours, diluted. (S. Solis-Cohen).

Cardiac complications are to a large extent prevented by the use of ammonium bromid, gr. xv-xx (1-1.3 Gm.), or ammonium salicylate, gr. x-xv (0.6-1 Gm.), in solution every four hours.

Subacute attacks and lingering cases are favorably influenced by salicylate of cinchonidia, gr. v (0.3 Gm.), every four hours, or—

℞.	Lithii salicylatis	gr. xv-xx	1.-1.3 Gm.
	Syr. zingiberis	fʒ j	4. Cc.
	Aq. lauro-cerasi	fʒ j	4. Cc.

M. S.—Every four hours.

Or—

℞.	Potassii iodidi..........	℈ iv	5.3 Gm.
	Sodii salicylatis	ʒ iv	15. Gm.
	Elix. cinchonæ	f℥ iss	45. Cc.
	Infus. gentianæ	f℥ iss	45. Cc.
	Aquæ destil.	f℥ j	30. Cc.

M. S.—Dessertspoonful every three or four hours, diluted.

These cases also do well with the use of salol, gr. v-x (0.3-0.6 Gm.), ammonium hydrochlorate, gr. xv-xx (1-1.3 Gm.), or salipyrin in solution every four hours.

℞.	Salipyrin	ʒ iij	12. Gm.
	Glycerini	fʒ iij	12. Cc.
	Syr. aurantii	fʒ vj	24. Cc.
	Aquæ destil.	ad f℥ vj	ad 180. Cc.

M. S.—Tablespoonful, well-diluted.

In all cases quinin sulphate, gr. xv (1 Gm.) daily, is of great value especially when there is hyperpyrexia, under which circumstance it should be administered hypodermically and accompanied by a cold bath or wet pack. The pains will be relieved to some extent by the coal-tar products but the best results will be obtained from the use of opium in some form, or atropin sulphate, gr. 1-80 (0.0008 Gm.) hypodermically, alone or combined with morphin.

Local Treatment.—Rest of the affected joint is essential. The inflamed parts should be wrapped in cotton-wool or flannel saturated with lead-water (two parts) and laudanum (one part), oil of gaultheria, fʒ i (4 Cc.), and compound soap liniment, f℥ iii (90 Cc.), or—

℞.	Sodii bicarbonatis	℥ ij	60. Gm.
	Tinct. opii	f℥ ss	15. Cc.
	Aquæ bul.	Oij	960. Cc.

M. S.—Use locally as directed.

The application of blisters, the size of a silver dollar, around the joint is very efficacious in relieving the pain and lessening the inflammation. If the joint condition tends to persist, equal parts of mercurial ointment and the ointment of belladonna will be found of great value. Baking of the joint in a hot-air apparatus is also beneficial. When the acute symptoms have subsided massage may be employed.

CHRONIC ARTICULAR RHEUMATISM.

Causes.—The affection may follow an acute or subacute attack, but in most cases it is chronic from the very beginning. It is observed usually in the poor, past middle life, and is influenced greatly by continued exposure to cold and wet and by heredity.

Pathologic Anatomy.—Thickening of the capsule and ligaments of the joints and the adjacent fibrous structures is a marked feature. In some cases the cartilages are eroded. Muscular atrophy and neuritis are observed as the condition progresses. These changes result in impairment of motion and false ankylosis.

Symptoms.—The principal symptoms are pain and stiffness in the joints aggravated by stormy weather. Tenderness and slight swelling may be present during the exacerbations. In most cases the condition is polyarticular. As the disease progresses the joint-movement may be seriously impaired or even lost entirely and the joints greatly distorted. Except in cases attended by severe pain of long duration, the general health may not be seriously impaired. The affection resists treatment and tends to persist indefinitely but does not endanger life.

Treatment.—The symptoms may be relieved to a great extent by judicious treatment but there is no curative treatment. Iodid of potassium, quaiac, iron, arsenic, and similar tonics should be administered. Residence in a dry and warm climate is particularly beneficial. The Turkish bath and bathing in the hot alkaline waters (Hot Springs of Virginia or Arkansas) are valuable.

Locally, counterirritation by means of the Paquelin cautery, or blisters, massage, electricity, and hot-air baths are very useful. Rubefacient liniments and absorbent ointments may also be employed.

MUSCULAR RHEUMATISM.

Synonyms.—According to location: cephalodynia; lumbago; torticollis; pleurodynia.

Definition.—An affection of the voluntary muscles, inflammatory in character, either acute or chronic; characterized by pain, tenderness, and stiffness of the affected muscles. It is never complicated with cardiac disease.

Causes.—A disease of adult life. One attack predisposes to another. Almost always due to cold or damp, or direct draught of cold air. Gout increases the tendency to attacks.

Pathologic Anatomy.—The true nature of muscular rheumatism is not yet determined. Virchow suggests a "hyperemia of, and scanty serous exudation between, the muscular striæ, and in chronic cases inflammatory proliferation of the connective-tissue."

Symptoms.—The first attack is generally acute, and its onset is rather sudden with pain, slight tenderness, and stiffness of the affected muscles, increased by any attempt at movement. These symptoms may be constant or may only be brought out on motion. Spasmodic contraction and rigidity of the muscles may be present. Fever is absent and there are no objective symptoms. The acute form seldom lasts more than a week; the chronic variety recurs frequently especially with changes in the weather, and may become constant.

Varieties.—It may affect any or all of the voluntary muscles, but its most frequent and important varieties are:

1. *Cephalodynia.* Situated in the occipito-frontal muscles. Distinguished from neuralgia of the trifacial, or occipital nerve, by pain on both sides of the head, excited or aggravated by the movements of the muscles and by absence of disseminated points of tenderness. The muscles of the eye may be affected, and movements of that organ excite pain. If the temporal and masseter muscles are attacked, mastication induces pain.

2. *Torticollis.* Wry neck, or stiff neck. Situated in the sternomastoid muscles. Generally limited to one side of the neck, toward which side the head is twisted, great pain being excited on attempting to turn to the opposite side. Rheumatism of the muscles of the back of the neck, *cervicodynia,* may be mistaken for occipital neuralgia.

3. *Pleurodynia.* Situated in the thoracic muscles, and may be mistaken for pleuritis, or intercostal neuralgia, from which it is differentiated by the absence of the diagnostic features of each. Pain is excited by forced breathing, coughing, and sneezing.

4. *Lumbodynia* or *lumbago.* Situated in the mass of muscles and fasciæ, which occupy the lumbar region. Most common variety. Usually affects both sides. It may set in rapidly, and become very severe. Motion of any kind aggravates the pain, often becoming very sharp or stabbing in character. It is sometimes complicated with acute sciatica, when the suffering is agonizing.

Prognosis.—Death never results from this condition. The attacks may be relieved by prompt and appropriate treatment but rheumatic tendency is often difficult to eradicate.

Treatment.—Rest is the first indication. This is accomplished in *pleurodynia* by firmly strapping the affected side with broad strips of plaster, extending from mid-spine to mid-sternum.

The local application to the affected muscles of hot poultices, made of two-thirds pilocarpus leaves and one-third flaxseed meal, changing them every two hours, is the most rapidly successful treatment in acute cases.

In all cases, the internal administration of antipyrin, gr. x-xx (0.6-1.3 Gm.), ammonium hydrochlorate, gr. xv-xx (1-1.3 Gm.), sodium salicylate, gr. xv-xx (1-1.3 Gm.), or lithium bromid, gr. v-xxx (0.324-1.944 Gm.), every three hours is of great benefit. When there is great pain and consequent insomnia the following should be used:

℞. Pulv. ipecac. et opii......gr. x .6 Gm.
Potass. nitrat. ....gr. v-x .3-.6 Gm.
M. S.—In powder, morning and night.

or morphin sulphate, gr. ⅛-¼ (0.008-0.016 Gm.), and atropin sulphate, gr. 1-80 (0.0008 Gm.), should be injected directly into the affected muscle and repeated as the occasion requires. When the disease is limited to a few muscles the following liniment is valuable:

℞. Ol gaultheriæ ....ʒ iss 6. Cc.
Spirit. vini rectif. ......f℥ ij 60. Cc.
M. S.—Thoroughly rub into affected part.

In all forms, but more particularly in lumbago, a few dry cups or blisters over the seat of pain will afford immediate relief. Other measures of value are hot-air baths, massage, electricity, and acupuncture.

In *chronic cases,* the administration of potassium iodid, guaiac, sulphur, ammonium hydrochlorate, arsenic, or gelsemium in various combinations is recommended. The bowels should be kept regular preferably by the use of salines. The local treatment is similar to that of the acute form being modified to suit the individual case.

RHEUMATOID ARTHRITIS.

Synonyms.—Arthritis deformans; rheumatic gout (?).

Definition.—A destructive disease of the joints, attended by osseous formations about the articulations, loss of motion, and deformity.

Causes.—The etiology is doubtful. It occurs most often in middle-aged women. Among the predisposing causes may be mentioned heredity, bad hygiene, exposure, injury, prolonged lactation, frequent pregnancies, menopause, grief, mental anxiety, tuberculosis, and frequent attacks of acute articular rheumatism. It is considered to be trophoneurosis by some observers and by others to be of infectious origin.

Pathologic Anatomy.—At first the affection is attended by hyperemia of the synovial membrane and increase of the synovial fluid. This is followed by proliferation of its cells with the subsequent formation of villous or nodular outgrowths. The capsular membrane becomes irregularly thickened and the synovial fluid decreases. As the process progresses the internal ligament is destroyed thus permitting dislocation. The interarticular fibrocartilages become ulcerated and disappear as do the cartilages covering the ends of the bones, thus exposing the articular extremities of the bones which become smooth, eburnated, and greatly enlarged. The villous nodules become ossified and the periosteum forms new bone. The adjacent ligamentous and fibrous structures become greatly thickened. Stiffness and impairment of motion are produced at first but later ankylosis (false), immobility, and deformities result. The surrounding muscles atrophy and neuritis is not infrequent.

Symptoms.—The affection may be acute or chronic. In the acute variety several joints are attacked at the same time and slight pyrexia is present. The affected joints are swollen and painful but other acute inflammatory phenomena are absent. The attack subsides more or less to recur after a varying interval.

The chronic form usually attacks but one joint at first, beginning, as a rule, in one of the metacarpophalangeal articulations. The joint slowly enlarges and becomes painful, neuralgic pains being excited by any attempts at movement. As the disease progresses the wrists, ankles, elbows, knees, jaws, and spine are involved, the corresponding joints on each side of the body being affected simultaneously. Movement is greatly impaired and soon the articulations become rigid. Crepitation is distinct after ulceration has destroyed the cartilage. Redness and tenderness are absent but swelling is marked. The muscles waste, thereby giving the joints the appearance of greater hypertrophy. Deformity soon manifests itself due to the disappearance of the cartilages and to contractures of the muscles. The fingers are bent backwards and drawn towards the ulnar side. The patient lies with the thighs and legs drawn up in adduction. Occasionally there is effusion into the joints. In addition to pain there may be tingling, numbness, local sweating, and pigmentation of the skin. The disease tends to advance slowly, ultimately involving all the joints and rendering the patient a helpless invalid.

Heberden's nodosities are the nodules, encountered in this disease, on the sides and ends of the distal phalanges of the fingers and toes. They occur most often in middle-aged women. They may be the seat of pain and tenderness, especially when the parts are cold or injured. Subjects having these nodules seldom have invasion of the large joints. Similar nodules are sometimes observed in gout.

Diagnosis.—*Chronic articular rheumatism* is often confounded with rheumatoid arthritis; but the former lacks the marked structural changes and the progressive involvement of joint after joint.

Gout differs from rheumatoid arthritis by the presence of deposits of urate of sodium in the joints, the ears, tips of fingers, and the bursæ over the olecranon process of the elbow, the presence of uric acid in the blood, and the decided history of acute paroxysms.

Gonorrheal rheumatism, so-called, has symptoms akin to rheumatoid arthritis, but the history of urethral discharge clears up the diagnosis.

Paralysis agitans, when pronounced, might be confounded with rheumatoid arthritis if the examination were limited to the joints; but the whole history, such as the tremor, the gait, etc., should prevent error.

Prognosis.—If early treatment be instituted, the disease may be held in abeyance for several years. After pronounced structural changes have begun, the malady is incurable, although it may remain stationary for a long time.

Treatment.—The diet and hygiene should receive attention, and all measures tending to improve the general health should be employed. Before serious structural changes have occurred, the rubbing into the joint of cod-liver with the internal administration of effervescing lithium citrate, ʒ i (4 Gm.) three times daily and the following tonic mixture is of value:

℞.	Massæ ferri carbonat. ...gr. v	.3 Gm.
	Liquor. potass. arsenit. ..♏ v	.3 Cc.
	Vini xerici ....fʒ j	4. Cc.
	Aquæ distill. ....fʒ j	4. Cc.

M .S.—After meals, well-diluted.

The internal administration of guaiacol carbonate, gr. v-x (0.3-0.6 Gm.) three times daily together, with painting of the joints, when painful, with the following is at times very efficient:

℞. Guaiacol ....1 part.
Tinct. iodi ....6 parts.
M. S.—Paint over joints twice daily.

Iron, arsenic, salicylates, etc., may also be employed. Massage is often of value.

GOUT.

Synonyms.—Podagra, gout in the foot; chiragra, the hand; gonagra, the knee.

Definition.—A constitutional disease, usually inherited; characterized by the sudden occurrence of a paroxysm of severe pain and swelling in one of the smaller joints,—the great toe usually,—with the presence of uric acid in the blood, and the deposit of the urate of sodium in the structure of the joint.

Causes.—The attacks usually follow some dietetic indiscretion such as the overindulgence in malt liquors and sweet wines, excessive eating, and exposure. Lead-poisoning, nervous strain, sedentary habits, and slight injury are also causes. The tendency towards gout is usually inherited, and men are affected with greater frequency than women. In the inherited variety there are some manifestations early in life, but in the acquired form it seldom shows itself before the age of thirty-five.

Pathologic Anatomy.—The disease is believed to be brought about by an excess of uric acid in the blood, salts of which, particularly the urate of soda, are deposited in the structure of the small joints and tissues that are not very vascular. As these deposits increase inflammatory reaction—hyperemia, redness, swelling, and effusion are produced, terminating ultimately in ulceration and expulsion of chalk-like masses of varying size. In many cases the deposits are retained for an indefinite period, becoming greater with each attack, finally causing great deformity and stiffness. The metatarsophalangeal joint of the great toe is usually first affected, but the deposits also accumulate in the knuckles, eyelids, and cartilages of the ear. Crystals of urate of soda may be seen by the naked eye in the tubules and intratubular structure of the kidneys, which become small, granular, and fibrous (gouty kidney). Hypertrophy of the left ventricle and arteriosclerosis usually accompany or result from this condition.

Symptoms.—*Acute gout* is rare in the United States. It occurs in paroxysms between which are varying intervals. The paroxysm is usually preceded, for a few days, by acid dyspepsia, constipation, headache, lassitude, insomnia, irritability of temper, etc. The attack in most cases begins suddenly, between midnight and two A. M. with acute pain in the ball of the great toe, which becomes red, hot, swollen, and so sensitive that the slightest touch cannot be borne. The veins are filled, the foot, ankle, and leg swollen, and the limb the seat of sudden spasmodic contractions, which increase the suffering; slight relief is afforded by elevating the limb. Associated with the local symptoms are chill, fever, quickened pulse, thirst, coated tongue, constipation, and scanty, acid, high-colored urine, which deposits, on cooling, a heavy brickdust sediment. Towards daylight the symptoms ameliorate, to return again at sundown, the severity gradually lessening, until the fourth or fifth day, when convalescence is established, the patient, as a rule, feeling better than before the attack.

Retrocedent gout is the term used to indicate those cases in which the symptoms suddenly disappear in the joints and are followed by alarming gastric, cardiac, or cerebral manifestations.

Chronic gout results from a repetition of acute attacks, and in typical cases the deposits may be detected in the various regions in which they are prone to occur with stiffness and varying grades of deformity in the smaller joints. Constitutional symptoms are present, but to a milder degree. Paroxysms are apt to occur at any time, but develop slowly with less pronounced symptoms.

Diagnosis.—The history, mode of life, age, acute onset, location, and the presence of the deposits (tophi) will serve to distinguish the affection from acute articular rheumatism and rheumatoid arthritis, with which it may at times be confounded.

Prognosis.—*Acute gout* is rarely fatal, but is prone to recur. Chronic gout is less favorable. The kidney, arterial, and cardiac complications materially shorten life. Acute diseases or injuries occurring in the course of chronic gout are more serious than under other circumstances.

Treatment.—In the *acute attack*, wine of colchicum root, gtt. xv-xxx (1-2 Cc.), well-diluted, should be given immediately and repeated every two hours until relief is afforded or the physiologic limit reached. It may be combined with sodium salicylate, gr. xx (1.3 Gm.), every two hours. The bowels should be opened by the administration of a course of calomel, followed by a saline. Water, especially the alkaline waters, should be freely consumed. The diet should be liquid, preferably milk. Bartholow recommends the following pill:

℞. Colchicinæ	gr. 1-40	.0013 Gm.
Ext. colocynth. comp.	gr. ss	.032 Gm.
Quininæ sulph.	gr. iij	.2 Gm.

M.—Ft. pil. No. i.

S.—One such pill to be taken every three hours.

The pain will call for the use of the coal-tar products and morphin, but the latter should be used with caution on account of the possible kidney complications. The affected part should be elevated and dressed with cloths soaked in lead-water and laudanum.

For *subacute* or *lingering cases* potassium iodid, alone or in combination, as the following is of value:

℞. Potassii iodidi	ʒ ij	8. Gm.
Vini colchici radicis	fʒ iv	15. Cc.
Aquæ destil.	f℥ iiss	75. Cc.

M. S.—Teaspoonful, well-diluted, after meals and at bedtime.

In *chronic gout* the diet, habits, mode of life, etc., should be subjected to considerable regulation. As far as practicable all nitrogenous or albuminous substances should be interdicted. The patient should be forbidden to partake of pastry, hot bread, cakes, sweetmeats, spices, condiments, veal, liver, mutton, lamb, pork, cheese, tomatoes, beans, oatmeal, sugar, tea, coffee, wines, and malt liquors. The foods permissible include milk, butter, oysters, fish, vegetables, and acid fruits, such as strawberries, lemons, and oranges.

The free use of water, particularly alkaline waters, such as Buffalo lithia, Farmville lithia, or Saratoga Vichy, should be encouraged. The administration of effervescing citrate of lithia, ʒi (4 Gm.), in water three times daily, and of the saline laxatives is also of value. The underclothing should be woolen, and it is advisable for the patient to seek a residence in a warm climate. Exercise and massage are of great importance in the treatment. Hydrotherapeutic measures—cold bathing, Turkish bath, etc.—when cautiously employed, are of great value.

The medicinal treatment includes the use of the alkaline carbonates, colchicum, salicylates, potassium iodid, guaiac, and tonics, such as iron, quinin, strychnin, and arsenic.

LITHEMIA.

Synonyms.—Lithiasis; uric acid diathesis; uricemia; American gout.

Definition.—A condition to which the fluids of the body are saturated with nitrogenized waste, in the form of lithic or uric acid; characterized by marked dyspepsia, various nervous phenomena, muscular and articular pains, bronchial catarrh, all or any of these associated with scanty, high-colored, acid urine.

Causes.—High living, with little exercise, sedentary habits, imperfect digestion of nitrogenous food, and impaired elimination of uric acid are the principal causes. The direct or remote offspring of gouty individuals are the most frequent subjects.

Pathology.—The pathology is not well-defined. The affection is believed to be due to faulty elimination of certain waste products which have a deleterious influence on the nervous system.

Symptoms.—Coated tongue, fetid breath, more or less loss or alteration in the appetite, dyspepsia, flatulence, abdominal distress, and constipation are common manifestations. The urine is scanty, high-colored, and acid in reaction. The specific gravity is about 1024-1028. Sugar and albumin are absent, but urates, uric acid crystals, and oxalates are present in large quantities. The urine is irritating and gives rise to pains in the loins and burning sensation in the urethra during its passage. Various nervous symptoms may be encountered, such as depression of spirits, impaired memory, sleeplessness, vertigo, headache, neuralgia, failure of memory, irritability of temper, ringing in the ears, and a constant dread of apoplexy or other cerebral diseases. Neuralgic pains in the joints, in the dorso-lumbar region, and in the right scapular region, are common. The second aortic sound becomes accentuated, and there is increased arterial tension. Various cutaneous affections, urticaria, erythema multiforme, erythema nodosum, etc., may occur as manifestations of this condition.

Complications.—When the affection persists for a long period untreated, chronic interstitial nephritis, fatty heart, enlargement of the liver, arteriosclerosis, chronic bronchitis, or angina pectoris may supervene as complications.

Diagnosis.—The history, the course, and the absence of well-marked joint symptoms serve to distinguish it from gout and rheumatism.

Prognosis.—The disease is essentially chronic, but when properly treated recovery is to be expected, otherwise the affection becomes serious through its complications.

Treatment.—Regulation of the diet is the first indication. The quantity of food consumed should be greatly reduced. Fresh meat should be eaten but once daily, and the diet list should be made up largely of milk, skimmed milk, milk and cream, poultry, game (plainly cooked), fresh fish, oysters, occasionally eggs, lettuce, spinach, celery, cold slaw, and tomatoes. All kinds of starchy and saccharine foods, fats, stimulants, tea, and coffee should be interdicted. Systematic exercise is of great importance. Water, especially the alkaline mineral waters, should be freely employed. Care should be taken to avoid the use of tonics, bromids, chloral, or opium in the treatment. Saline laxatives should be administered, followed by intestinal antiseptics, such as salol, gr. v (0.32 Gm.), three times daily, or potassium permanganate, gr. i (0.065 Gm.), after meals.

Good results follow the use of lithium citrate, gr. xx (1.3 Gm.) three times daily, sodium phosphate, gr. xxx-lx (2-4 Gm.) three times daily, or benzoic acid, gr. x (0.6 Gm.) three times daily. One of the best drugs in this condition is dilute nitric acid, ♏x (0.6 Cc.) in half a glass of water four times daily, with the occasional use of compound rhubarb pill at bed-time. Considerable benefit is at times derived from piperazin, gr. x-xv (0.6-1 Gm.) in solution after meals, and also from strontium in combination:

℞. Strontii bromidi puræ ....gr. xxx	2. Gm.
Glycerini ....♏xxx	2. Cc.
Infus. gentianæ ....fʒ iss	6. Cc.

M. S.—Before meals, well-diluted.

RACHITIS.

Synonym.—Rickets.

Definition.—A nutritional disease occurring in early childhood characterized by changes in structure of the bones, with consequent deformity, muscular weakness, and nervous disturbances.

Causes.—The affection usually develops in the early months of the second year, although in rare instances it may be congenital. Among the important etiologic factors may be mentioned negro race, foul air, insufficient or improper food, lack of sunlight, dampness, poverty, and city life. Syphilis may be a cause in some cases.

Pathologic Anatomy.—The structural changes are most marked in the bones of the skull, the long bones, and the ribs. The head assumes a box-shape, due to enlargement of the parietal and frontal eminences and flattening of the occiput and top of the head. The fontanels often remain open until the second or third year. In the long bones, the cartilages between the epiphysis and shaft become swollen and spongy in structure. The periosteum is thickened and by its proliferation spongy tissue is also formed. The affected parts show a marked deficiency in lime salts. The bones are consequently soft and bend easily, giving rise to deformities such as bow-legs, knock-knees, pigeon-breast, curvature of the spine, etc. Green-stick fractures are not uncommon results. The chondral ends of the ribs become enlarged and nodular, giving the breast a beaded appearance (rachitic rosary). Chemical examination of the diseased bones shows an increase in the percentage of organic matter with a marked diminution in the proportion of inorganic or mineral constituents. The liver, spleen, and sometimes the mesenteric glands are enlarged.

Symptoms.—In addition to the various changes in shape in the head, chest, and long bones, there may also be present restlessness and feverishness at night, with profuse perspiration about the head, diffuse tenderness, nausea, vomiting, abdominal distention, slight diarrhea, nervousness, convulsions, etc. Dentition is delayed and when the teeth do appear they are badly formed. Muscular weakness is marked and prevents the child from walking or even sitting up (pseudoparalysis).

Complications.—The profound weakness predisposes to all the various affections of childhood. The most common complications are bronchial catarrh, bronchopneumonia, atelectasis, chronic hydrocephalus, diarrhea, laryngismus stridulus, and convulsions.

Prognosis.—The disease is not fatal in itself, but may become serious in the presence of complications. Deformities are common sequels, and in the case of the female pelvis may be of grave importance in subsequent labors.

Treatment.—The first indications are to place the child in hygienic surroundings and to provide proper food. If the child is nursing and the mother's milk is poor, cow's milk should be substituted and properly modified to suit the individual requirements. Older children should be given beef-juice, eggs, and beef peptonoids, in addition to milk. Starches and sugars should be avoided. Orange and lemon juice are beneficial in many cases. Thin gruels may be used. Cod-liver oil, syrup of iodid of iron, hypophosphite of calcium, lactophosphate of calcium, lime-water, and phosphorous, are the drugs usually employed in this condition.

DIABETES MELLITUS.

Synonyms.—Glycosuria; melituria.

Definition.—A chronic affection characterized by the constant presence of grape-sugar in the urine, an excessive urinary discharge, and the progressive loss of flesh and strength.

Causes.—The affection is most commonly observed in males, most often in Hebrews between the ages of twenty-five and fifty years. It is rare in negroes. Among the most important etiologic factors may be mentioned inherited tendency, disorders of the nervous, hepatic, and renal systems, excessive use of farinaceous foods and malt liquors, sedentary habits, mental anxiety, and sexual excesses.

Pathology.—The disease is believed to be due primarily to some disturbance of the nervous system. Experimental puncture of the floor of the fourth ventricle has produced it, as has also disease and extirpation of the pancreas. In a large proportion of cases it is possible to demonstrate changes in the pancreas, but more frequently hyperemia and hypertrophy, sometimes degeneration, of the liver and kidneys may be observed. The pathogenesis is extremely obscure. There are no constant lesions.

Symptoms.—Clinically, cases differ greatly in their course and severity; one class presenting slight symptoms and a chronic course; another class having marked local and constitutional symptoms and running an acute course. The symptoms of a typical case may be arranged under the following heads:

Urinary Symptoms.—Micturition is frequent and accompanied by pain in the region of the kidneys. The urine is greatly increased in quantity (4, 8, 12, 20, or even 30 pints in 24 hours). It is pale, clear, and watery, having a sweetish taste and odor. The specific gravity ranges from 1025 to 1050. It ferments rapidly if kept in a warm place. Sugar is present in amounts varying from an ounce to two pounds in twenty-four hours. The urea and uric acid are increased. Albumin may be present. The increased passage of a large quantity of saccharine urine causes a constant itching, burning, and uneasy sensation at the prepuce, along the urethra, and at the neck of the bladder; in females, itching and eczema of the vulva are common; in children, incontinence of urine is frequent.

Digestive Symptoms.—Thirst is almost constant, and the mouth is dry and parched. The breath may have a sweetish odor and the tongue is irritable, beefy red, and often cracked. The appetite is variable, at times excessive, at others, absent. Vomiting occasionally occurs. Dyspeptic symptoms are common. Constipation, with pale and dry stools, is the rule, but diarrhea may occur.

General Symptoms.—The patient complains of feeling very weak and languid, and of soreness and pains in the limbs. Emaciation soon becomes marked. The skin is harsh, dry, and often intensely itchy. The countenance assumes a distressed and worn expression. Various nervous phenomena make their appearance. The mind is often greatly altered; depression of spirits; decline in firmness of character and moral tone; and irritability are present. Neuralgia and headache are common. Sexual inclination and power are greatly diminished. Visual defects are not infrequent. The temperature is usually below normal. The heart's action is weak, with a frequent low-tension pulse. The blood and various secretions contain sugar.

Complications.—The principal cutaneous complications are boils, carbuncles, pruritus, eczema, and gangrene, especially of the feet and legs. The pulmonary complications of greatest frequency are tuberculosis, lobar pneumonia, and gangrene. The most common eye complications are cataract, retinitis, optic atrophy, palsies, and toxic amblyopia. The nervous complications include peripheral neuritis, ringing in the ears, deafness, and diabetic coma or acetonemia, a condition characterized by unconsciousness, dyspnea, pain in the head, delirum, rapid and feeble pulse, sweetish odor of the breath, and the presence of acetone in the urine. Nephritis may also occur as a complication.

Course.—In most instances the course is chronic, lasting for years, the symptoms beginning insidiously and becoming progressively worse, with, at times, decided remissions. Occasionally the disease runs an acute course, death occurring within four or five weeks.

Diagnosis.—Diabetes mellitus only exists when grape-sugar is permanently present in the urine. "It is not the quantity, but the persistence of sugar which constitutes diabetes." With grape-sugar in the urine, associated with more or less increase in the urinary flow, it should be mistaken for no other affection.

It may distinguished from *Bright's disease,* by the absence of dropsy, and of tube-casts in the urine, and the constant presence of sugar in the urine; but the amount of albumin in the urine is never so great or constant in diabetes mellitus as in Bright's disease.

From *diabetes insipidus,* it may be separated by the presence of sugar in the blood and urine, and by the larger quantity of urine voided in polyuria.

Simple glycosuria differs from diabetic glycosuria in that the amount of sugar in the urine is not constant,—at one time being present, at another absent,—the amount of urine voided is never in excess of health; simple glycosuria is a disease of the aged; diabetic glycosuria usually appears under fifty years. Simple glycosuria often results from the inhalation of chloroform, the excessive use of chloral, and as one of the results of injuries to the head. It may also occur from excitement and in the insane.

Prognosis.—The majority of cases ultimately prove fatal from gradual exhaustion or from profound blood-poisoning, ending in diabetic coma, or, rarely, uremia. The complications are often the direct cause of death. Amelioration of the symptoms may occur and the progress of the malady may be greatly retarded with treatment. Complete recovery seldom, if ever, occurs. The younger the patient the more rapid is the course of the disease. Surgical operations should not be undertaken in diabetic patients on account of the tendency to gangrene.

Treatment.—The treatment of diabetes may be conveniently considered under three headings: dietetic, hygienic, and medicinal.

Dietetic Treatment.—The diet should be so regulated as to exclude or at least to reduce to a minimum the quantity of starches and sugars. The patient should be allowed to partake of meats of every kind, soups made with meat and without flour, game, poultry, fish, oysters, lobsters, crabs, eggs, butter, cheese, oils, fats, cream, buttermilk, milk, spinach, celery, lettuce, cabbage, tomatoes, asparagus tops, water-cress, string-beans, onions, cucumbers, pickles, olives, unsweetened jellies, almonds, walnuts, butternuts, filberts, apples, lemons, strawberries, tea and coffee without sugar, claret, Burgundy, and Rhine wines, carbonated waters, and bread made from gluten, bran, or almond flour.

The substances which should be especially denied the patient are ordinary bread or flour, sugar, honey, potatoes, parsnips, peas, barley, beans, rice, tapioca, arrowroot, cracked wheat, oatmeal, turnips, beets, corn, carrots, prunes, grapes, figs, bananas, pears, peaches, watermelons, canteloupes, chestnuts, chocolate, biscuits, pastry, syrups, preserves, sweet wines, and malt liquors.

When sweetening of the food is absolutely necessary, saccharin or glycerin may be used for that purpose.

Hygienic Treatment.—Fresh air, daily bathing, and regular exercise are essential to the treatment. Perfect ventilation of the apartment in which the patient works and sleeps is highly important. The use of various mineral waters is very beneficial. The exercise should be taken daily and regulated according to the patient's strength, being careful to avoid over-exertion. Flannel underclothing should be constantly worn.

Medicinal Treatment.—A great number of drugs are recommended for this condition, most of which are useless. The most valuable drug is opium or one of its derivatives. Codein, gr. ss-iii (0.032-0.2 Gm.), three times daily, gradually increasing the dose, is the alkaloid most commonly employed. Morphin hydrochlorate, gr. i (0.065 Gm.) daily, or powdered opium, gr. iii-v (0.2-0.3 Gm.) daily, may be used instead. The constipation which these preparations are prone to produce should be combatted by the use of the natural aperient waters, such as Hunyadi, Carlsbad, Vichy, etc. Arsenic, in the form of Fowler's solution, is especially valuable in this disease. Ergot and ammonium carbonate are also employed at times with benefit. Uranium nitrate, gr. iii (0.2 Gm.) three times daily, and sodium salicylate, gr. xv (1. Gm.) three times daily, will often lessen the quantity of urinary secretion and reduce the amount of sugar. The bromids are efficient in controlling the nervous symptoms; potassium or sodium bromid, ʒi (4 Gm.) in twenty-four hours, or the solution of the bromid of arsenic, ♏iii-v (0.2-0.3 Cc.) three times daily, may be employed. The alkalies, and especially the alkaline carbonates and alkaline mineral waters, are of especial value. The coal-tar products, antipyrin, antifebrin, and phenacetin, in doses of gr. x-xv (0.6-1 Gm.) three times daily, combined with an equal quantity of sodium bicarbonate will be found very sufficient in some cases of mild type. Powdered jambul seeds, gr. v-x (0.3-0.6 Gm.) three times daily, and methylene blue, gr. viii (0.52 Gm.) daily, have been used with success. Among other drugs used in this condition may be mentioned pepsin, iodin, potassium iodid, lactic acid, glycerin, quinin, tincture of cannabis indica, cod-liver oil, and adrenalin. The galvanic current is sometimes of value. The emaciation calls for the use of tonics in addition to the remedies employed to combat the disease. The sleeplessness and unrest require morphin, bromids, chloral, or hyoscin hydrobromate. The excessive thirst may be greatly relieved by the following:

℞. Potassii phosphat. ......two parts.
Aquæ ....seventy-five parts.

M. S.—One teaspoonful twice or thrice daily, in wine or hop tea.

Diabetic coma requires inhalations of oxygen, subcutaneous injections of sodium carbonate, and hypodermoclysis and enteroclysis. The alkaline treatment is regarded as a preventive measure in this complication.

DIABETES INSIPIDUS.

Synonyms.—Polyuria; polydipsia.

Definition.—An affection characterized by the excessive secretion of a very large quantity of pale, watery urine, free from albumin and sugar.

Causes.—The affection may be inherited or diabetes mellitus may have existed in the parent. It is most often observed in children and young adults. Men are more liable than women. Injuries, tumors, and diseases of the nervous system, hysteria, exposure to cold, consumption of excessive quantities of cold water, fatigue, prolonged debility, malaria, syphilis, and intense emotional excitement may produce the condition. The probable immediate cause of the excessive secretion of urine consists in dilatation of the renal vessels, the result of paralysis of their muscular coat, caused by derangement of innervation, since the condition can be induced experimentally by irritating a certain area in the fourth ventricle, or by section of portions of the sympathetic nerve.

Symptoms.—The affection is characterized by great thirst, with an increased flow of pale, watery, slightly acid urine, the amount varying from one to five or six gallons in the twenty-four hours. The specific gravity ranges from 1.001-1.007. Sugar and albumin are absent. Urea and the other solids are increased. The appetite is voracious, the bowels are obstinately constipated, and the skin is dry and harsh. The large flow of urine is usually preceded by various nervous phenomena, as nervousness, irritability, inability to concentrate the mind, vivid imagination, a failure of memory, and headache. Unless the affection is soon arrested, great loss of flesh and strength result.

Diagnosis.—It differs from *diabetes mellitus* by the absence of grape-sugar in the urine.

From *paroxysmal diuresis,* by the presence of the increased urine permanently.

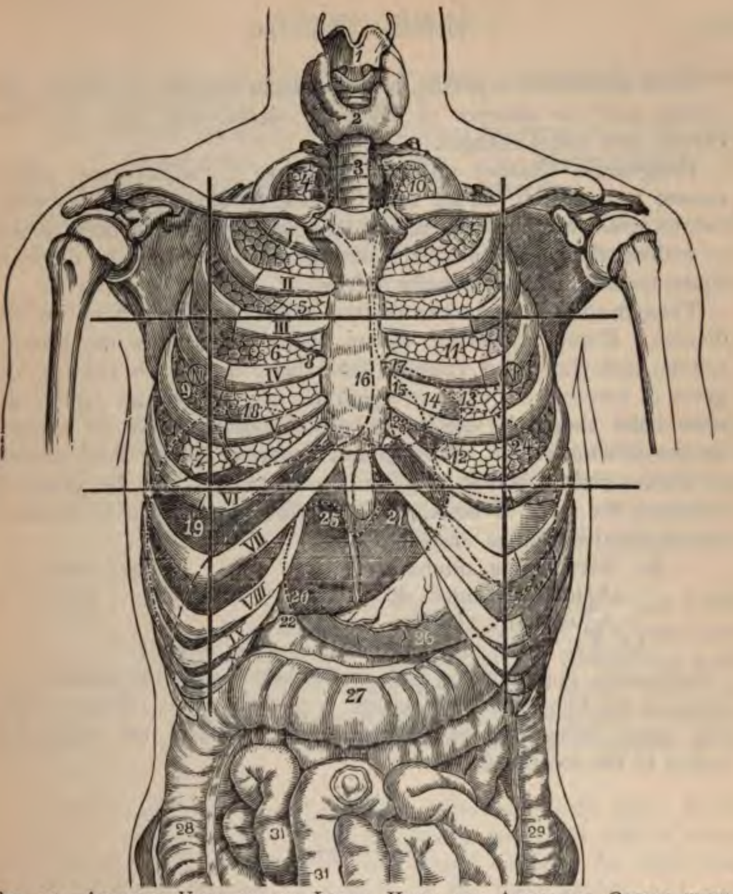

Fig. 25.—Anterior View of the Lungs, Heart and Abdominal Organs with Reference to their Relation to the Skeleton and the Outline of the Stomach. (*Tyson's Diagnosis.*)

1. Larynx. 2. Thyroid gland. 3. Trachea. 4. Right lung-apex. 5. Upper lobe. 6. Middle lobe. 7. Lower lobe, of right lung. 8. Upper, 9. Lower interlobular boundary of the right lung. 10. Apex. 11. Upper lobe. 12. Lingual process of the left lung. 13. Cardiac boundary of the anterior border of the left lung. 14. Portion of the anterior aspect of the pericardium covered by the cardiac pleura. 15. Portion of the same uncovered by diaphragm. Site for paracentesis. 16. Anterior border of the mediastinum. 17. Anterior border of the left mediastinum. 18. Upper or true border of the liver partially covered by lung. 19. Right lobe of the liver. 20. Quadrate lobe of the liver. 21. Left lobe of the liver. 22. Gall bladder. 23. Cardiac end of the stomach. 24. Stomach cul-de-sac partially covered by lung. 25. Pyloric end of the stomach. 26. Larger curvature of the stomach ((right gastro-epiploic artery). 27. Transverse colon. 28. Ascending colon. 29. Descending colon. 31. Small intestine.— (After *Luschka,* slightly modified.)

From *interstitial nephritis*, by the greater amount of urinary discharge and the absence of albumin, edema, and casts, and the cardiac and vessel changes.

Prognosis.—Rather unfavorable as to a radical cure, unless caused by syphilis. Death rarely is due to the diabetes insipidus, but to some intercurrent malady that the patient has been unable to withstand, on account of the weakness produced by the diabetes. Spontaneous cure occasionally occurs.

Treatment.—Restriction of the fluids has no effect on the disease. Ergot, pilocarpin, opium, gallic acid, potassium bromid, sodium salicylate, and valerian have been used with varying degrees of success. In cases of syphilitic origin, mercury and potassium iodid should be employed. Constipation should be avoided by the administration of compound cathartic pills. Tonics, such as iron, quinin, arsenic, strychnin, etc., should also be given to maintain the general health. The following formula is producttive of great benefit in this disease:

℞.	Strychninæ sulphatis ...gr. 1-48	.0015	Gm.
	Acid. hydrochlor. dil. ... ♏x	.6	Cc.
	Aquæ lauro-cerasi .....fʒ ij	8.	Cc.

M. S.—To be taken three times daily in water.

Galvanism, applying one pole to the neck below the occiput, the other to the hypochondriac region, is also of value. Warm clothing, warm baths, friction, fresh air, exercise, etc., are useful adjuncts to the treatment.

DISEASES OF THE RESPIRATORY SYSTEM.

PHYSICAL DIAGNOSIS.

Physical Diagnosis is the art of discriminating disease by means of the eye, the ear, and the touch.

The signs thus ascertained are connected with changes or alterations in the form, density, or condition of the structures within, and are known as physical signs.

"Physical signs are, then, the exponents of physical conditions, and of nothing more." (Da Costa.)

The methods employed in the physical exploration of the chest, are: I, **Inspection**; II, **Palpation**; III, **Mensuration**; IV, **Percussion**; V, **Auscultation**; VI, **Succussion.**

Chest Divisions.—For the purpose of physical exploration, the chest is mapped off into regions or divisions, as follows:

For the purpose of physical exploration, the chest is mapped off into regions or divisions, as follows:

ANTERIORLY.

First.—*Supra-clavicular,* Lying above the upper edge of the clavicle, usually about an inch in extent. It contains the apex of each lung, with portions of the subclavian and carotid arteries and the subclavian and jugular veins.

Second.—*Clavicular,* Corresponding to the inner two-thirds of the clavicle.

Third.—*Infra-clavicular,* From the clavicle to the lower border of the third rib, and from the edge of the sternum to a line drawn vertically downward from the junction of the middle and outer third of the clavicle. This region contains the upper lobe of the lung and the main bronchi; on the right side the superior vena cava and part of the aortic arch; and on the left side a portion of the pulmonary artery.

Fourth.—*Mammary,* Between the third and sixth ribs. In the center of this region between the fourth and fifth ribs is placed the nipple. On the right side this region contains the right lung, part of the diaphragm, a portion of the right auricle and right ventricle, on the left side, the lung and a small part of the right ventricle.

Fifth.—*Infra-mammary,* Downward from the sixth rib to the margin of the false ribs. On the right side it contains the liver and a small portion of the lung on deep inspiration; on the left side, the lobe of the liver, stomach, and part of the spleen.

Sixth.—The *upper sternal region* extends from the suprasternal notch to the junction of the third costal cartilage and sternum. The ascending arch of the aorta, portions of the superior vena cava, the innominate veins, subclavian arteries, esophagus, and trachea are found in this region.

Seventh.—The *lower sternal region* extends downward from the junction of the third costal cartilage with the sternum, and contains portions of the lung, right and left ventricles, and stomach.

The *mammary line extends* downward from the junction of the middle and outer third of the clavicle.

The *parasternal* line is a vertical line placed midway between the border of the sternum and the mammary line.

LATERALLY.

First.—*Axillary,* That portion above the sixth rib. The upper lobes of the lung and the main bronchi are to be found in this region.

Second.—*Infra-axillary,* That portion below the sixth rib. On the right side, it contains the lung and liver; on the left side, the lung, stomach, and spleen.

POSTERIORLY.

First.—*Supra-scapular,* That portion above the scapula.

Second.—*Scapular,* That portion covered by the scapula. It contains the greater portion of the lung.

Third.—*Inter-scapular,* That portion between the scapulæ. It extends from the second to the sixth vertebra and contains portions of the lungs, bronchi, esophagus, and descending aorta.

Fourth.—*Infra-scapular,* That portion below the angle of the scapula and above the twelfth rib. On the right side, it contains a portion of the lung, liver, and kidney; on the left side, a portion of the lung and intestine, spleen, kidney, and descending aorta.

INSPECTION.

Inspection signifies "the act of looking." Views of the chest should be taken from the sides and behind as well as from the front, for which purpose a good light should be obtained, and the patient be placed in as easy and comfortable a position as is possible.

THE CHEST AND ABDOMINAL CAVITY.

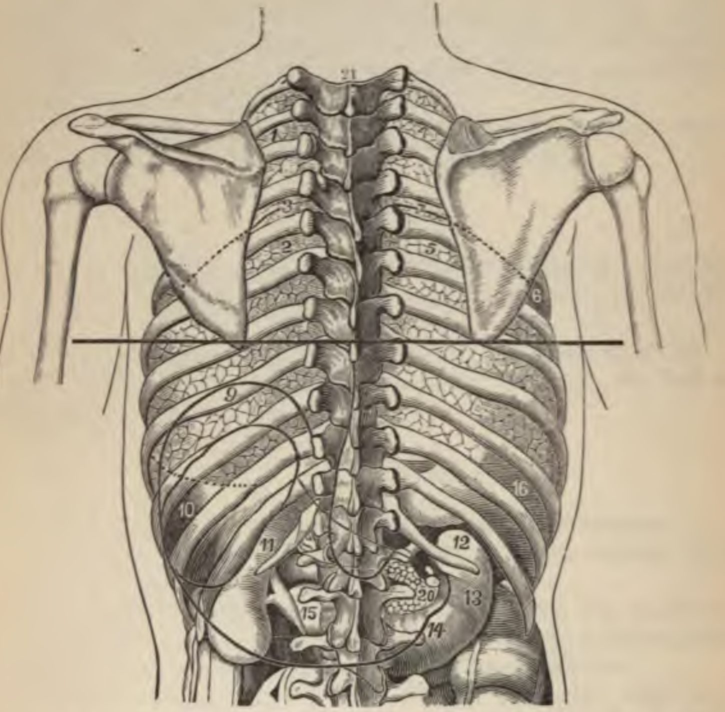

Fig. 26.—Posterior View of the Organs of the Chest and Abdominal Cavity. (*Tyson's Diagnosis.*)

1. Upper lobe. 2, Lower lobe of left lung. 3. Interlobular boundary between them. 4. Upper lobe of right lung. 5. Lower lobe of right lung. 6. Middle lobe of the right lung. 7. Line between upper and middle lobes of the right lung. 9. Stomach demarked by a dark line. 10. Spleen in its relation to the lung in expiration with the kidney showing behind and below it. 11. Left kidney. 12. Horizontal upper part of the duodenum. 13. Descending portion of the duodenum. 14. Horizontal lower part of the duodenum. 15. Duodeno-jejunal flexure. 16. Liver. 20. Pancreas. 21. First dorsal veretbra.—(*After Luschka.*)

Inspection reveals the *form, size, color,* and *movements* of the chest, as well as the condition of the superficial parts.

Variations in Form.—The *phthisinoid chest* is characterized by a short anteroposterior diameter and a long vertical diameter. The chest is flat, and the ribs are oblique. The supra and infra-clavicular spaces are much depressed and an acute angle is formed at the lower portion by the divergence of the costal margins from the sternum. Posteriorly the scapulae are very prominent.

The *rachitic chest* is marked by flattening of the sides, with prominence of the sternum (pigeon-breast). Beading of the sternal ends of the ribs (rachitic rosary) and circular constriction of the chest at the level of the xiphoid cartilage (Harrison's groove) are often present in addition.

The *emphysematous chest* is distinguished by being abnormally round and short. The transverse and anteroposterior diameters may be equal in some cases. It is also known as the "keg or barrel-shaped chest." The ribs are horizontal and the costal angle is obtuse or obliterated.

Sternal depressions are usually congenital, but may be acquired as the result of occupation, as in the case of shoemakers.

Sternal enlargements are generally congenital.

Localized depressions of the chest may result from pulmonary tuberculosis, fibroid lung, or adhesions following pleurisy or other pulmonic conditions.

Localized or one-sided enlargements may be caused by pleural effusion, compensatory emphysema, tumors of the abdomen or chest, pneumothorax, hydrothorax, hemothorax, and enlargement of the heart (left side).

Expansion.—In health the sides of the chest are for the most part symmetrical in form, size, color, and movements, both sides rising equally during the act of inspiration, and falling equally during the act of expiration. During the act of inspiration the intercostal spaces in the lower two-thirds of the chest become more hollow, as do also the supra-clavicular fossæ.

Inspiration is almost entirely the result of muscular action; *expiration,* on the other hand, is chiefly due to the elasticity of the lungs and chest-walls, aided somewhat in forced respiration by muscular action. The movement of inspiration by inspection is of longer duration than that of expiration, and the pause between the acts but momentary.

The *respiratory movement* is visible over the whole thorax, although in males and in children it is most distinct at the lower portion (*inferior costal breathing*), while in the female it is most distinct at the upper portion of the chest (*superior costal breathing*).

The *abnormal variations in expansion* of the chest are usually unilateral. Diminished unilateral expansion is common to acute pleurisy, pulmonic consolidation from any cause, collapse of the lung from bronchial obstruction, tumors, and abdominal enlargements. Increased unilateral expansion occurs in compensatory emphysema.

PALPATION.

By **palpation** is meant the application of the palmar surfaces of the hands and fingers to the chest, by means of which are appreciated impressions, that are capable of being conveyed by the sense of touch.

The objects of palpation are:

First.—To give more accurate information of what is revealed by inspection.

Second.—To locate areas of tenderness, the density and condition of tumors, if any be present; the state of the chest-walls, the frequency of the breathing, and the action of the heart. Tenderness of the chest-wall may be produced by traumatism, caries and fracture of the ribs, intercostal neuralgia, pleurodynia, and pleurisy (alone or combined with other lung conditions as phthisis and pneumonia). Edema of the chest may be due to anasarca, empyema, or pulmonary abscess.

Third.—To determine the existence and character of the various kinds of *fremitus* (vibrations). By fremitus is understood certain tactile impressions or vibrations conveyed to the surface of the chest, which are classed and produced as follows:

First.—*Vocal fremitus,* produced by the act of speaking or crying.

Second.—*Tussive fremitus,* produced by the act of coughing; of value especially when the voice is very weak.

Third.—Bronchial fremitus, produced by the passage of air through mucus, blood, or pus, in the bronchial tubes, during the act of respiration.

Fourth.—Friction fremitus, produced by the rubbing together of the roughened surfaces of the pleura.

When the normal chest vibrates lightly, it is termed the *normal vocal fremitus.*

The *vocal fremitus* is more distinct upon the right side toward the apex.

If the lung be consolidated (denser), the vibration is greater and more easily distinguished,—*the vocal fremitus is increased.* As examples of conditions with increased vocal fremitus, may be mentioned croupous pneumonia, phthisis, and bronchopneumonia.

In feeble persons, or when any cause interferes with the transmission of the vibrations, the *vocal fremitus is diminished* or absent. This is observed in pleural effusions, emphysema, collapse of the lung, tumors, and pulmonary edema.

MENSURATION.

Mensuration, or measurement of the chest, is of little practical importance, and hence seldom performed. The only measurement likely to be required is the circular or circumferential, in different parts of the chest; this is taken with either an ordinary graduated tape-measure or a double tape-measure, made by uniting two tapes in such a manner that they start in opposite directions from the same point at the *mid-spinal line.* The tapes drawn around each side until they meet at the *mid-sternal line,* on a line immediately above the nipple, or on the level of the sixth rib near its attachment to the cartilage,—the sixth costo-sternal joint,—the patient first being directed to effect a complete expiration, the number of inches noted, and then to take a deep inspiration, the increase in inches noted, the difference between the two giving a rough estimate of the capacity of the lungs.

In right-handed persons the right side is usually one-half to three-fourths of an inch larger than the left; if larger than this, it is usually the result of some abnormal condition.

In well-developed men, the chest measures at the upper part about thirty-three to thirty-six inches during expiration, and is increased fully three inches upon inspiration.

PERCUSSION.

Percussion, or "The act of striking," to ascertain the composition of structures, affords signs and information of great value in diagnosis.

There are two methods employed, *immediate* and *mediate.*

Immediate, or direct percussion, is performed by striking the thorax directly with the points of the fingers or the palmar surface of the hand. This method of percussion has been generally abandoned, as it does not enable the physician to distinguish, with sufficient accuracy between the various shades of difference in the pitch or quality of percussion sounds.

Mediate, or indirect percussion, may be practised in three different ways, to wit.:

First.—With the finger of one hand interposed between the body percussed and the percussing finger.

Second.—With the finger acting as a pleximeter and the percussion hammer.

Third.—With the percussion hammer and the pleximeter.

The first of these modes affords the most correct and ready information regarding the *resistance* of the parts percussed. The skillful use of the fingers is more difficult to acquire than that of the pleximeter and hammer; but if the examiner has acquired sufficient skill in its performance, an absolutely accurate result may be obtained. "He who is skilled in digital percussion will be able to percuss equally well with the hammer, the inverse of which does not always hold good." In addition to being proficient in technic, it is necessary to possess a sensitive ear, educated to distinguish between the various shades of the sounds.

When the fingers are employed, it is a matter of choice whether one or more fingers are used as the pleximeter. Usually the last phalanx of the first or second fingers of the left hand are used, the other fingers being *raised from the chest, so as not to interfere with the sound vibrations;* they should be applied *firmly* and *evenly* to the surface, thus preventing the slipping of the soft parts, and also to determine the resistance of the chest-walls when the blow is given. The *rounded ends* of the first and second fingers of the right hand are used as a hammer, striking the pleximeter fingers in such a manner that the nails shall not touch the skin of the underlying fingers. The force employed varies in different regions, but usually, for the chest, should be only of moderate degree. Forcible percussion is of use only when the sound of deep-seated organs is desired.

The *stroke* should be made perpendicularly to the surface, and not slanting, as is too often done. The whole movement should proceed only from the *wrist-joint,* and ought not to be too rapid or unequal, or of great force, the fingers being rapidly withdrawn, so as not to interfere with the vibrations.

The objects of percussion are to elicit certain *sounds*, and the amount of *resistance* or *elasticity* of the organs percussed.

The main sounds elicited by percussion are the *dull, clear,* and *tympanitic.* Familiarity with the *intensity, character,* and *pitch* of each of these sounds is essential.

When percussing the healthy chest, the sound obtained is termed the *normal pulmonary resonance.* It is of variable *intensity,* depending upon the force of the stroke employed and the amount of adipose and muscular tissues covering the thorax, and the *tension* of the chest-walls.

There is no exact standard of the normal pulmonary or vesicular resonance, but if the two sides of the chest are compared, the normal standard of each person is obtained.

The *character* is termed *pulmonary* or *clear,* as characteristic of the healthy chest-wall. The *pitch* is always relatively low.

The sounds elicited by percussing a healthy chest are not, however, alike over all its parts.

Anteriorly, the portion of lung above the clavicle yields a sound which becomes somewhat *tympanitic* as the trachea is approached.

Over the clavicle the sound is clear and pulmonary at the center of the bone, but at the scapular extremity it is duller, and toward the sternum it becomes somewhat tympanitic.

At the *infra-clavicular region* the resonance is clear and distinct, but little resistance being offered to the percussing finger, and the sound elicited may be taken as the type of the pulmonary resonance. In this region, however, a slight disparity exists between the two sides; on the right side the sound is less clear, shorter, and of a higher pitch than on the left side.

In the *mammary region* of the right side the resonance of the lung is not so clear, the sound being modified by the size of the mamma and the upper border of the liver. On the left side the heart deadens the sound from the fourth to the sixth rib, and, in a transverse direction, from the sternum to the left nipple. This dull sound in the left mammary region is lessened in extent during full inspiration, and in emphysema, when the lung more completely covers the heart.

In the *infra-mammary region* on the right side the percussion note is dull, except during the act of complete inspiration, when the liver is displaced downward by the inflated lung. In the left *infra-mammary region* the sound consists of a mixture of the dull sound of the heart and spleen and of the clear sound of the lung, together with the tympanitic sound of the stomach. In the lower part of this region is an area known as *Traube's semilunar space,* over which the note is tympanitic. It is bounded above by the sixth rib, on the left by the spleen, and on the right by the liver.

Over the upper part of the *sternum*—above the third rib—the sound is slightly tympanitic. Below the third rib, over the sternum, the sound is dull, due to the presence of the heart and liver.

The *position* exercises some influence on the results of percussion. More accurate results are obtained when the patient is standing or sitting than when recumbent. While the front of the chest is percussed, the arm should hang loosely by the sides; the hands may be clasped across the top of the head during the percussion of the axillary region; during the examination of the back the head must be bent forward and the arms tightly crossed in front.

On the *posterior* surface of the chest the sound also varies according to the part percussed.

Over the *scapulæ* the sound is duller than between these bones or below their inferior angles.

Over the *infra-scapular region* a clear sound is obtained as far as the lower border of the tenth rib on the right side, where the dullness of the liver begins. On the left side, below the angle of the scapula, the percussion sound is tympanitic if the intestines are distended, or it may be slightly dull if the spleen is enlarged.

In the *axillary* region the sound is clear and distinct on each side.

In the *infra-axillary* region of the right side the sound is duller, owing to the presence of the liver; at the corresponding situation on the left side the sound is clear or tympanitic, from the distention of the stomach, and at the ninth or tenth rib of the left axillary region, dullness and the sense of resistance mark the location of the spleen.

The sounds obtained by percussion of the unhealthy or abnormal chest are as follows:

First.—Hyper-resonance, or an increase of the normal pulmonary resonance, is due to the relative increase in the proportion of air to the solid tissues of the lung, provided the tension of the chest-walls be not altered. It occurs in emphysema of the lungs, atrophy of the lungs, or consolidation of the opposing lung.

Second.—Dullness or an absence of resonance, due to the relative increase of solid tissues in proportion to the amount of air, as seen in the different stages of phthisis, in pneumonia, pleural effusion, and hydrothorax.

The *pitch* is increased or heightened in proportion to the diminution of the amount of the air and the increase of the solids.

If there be entire want of resonance, the percussion note is said to be flat; if there is a slight decrease in the resonance of the part, the note is said to be impaired.

The sense of *resistance* is greater, the more marked the consolidation of the lungs and the greater the tension of the chest-walls.

Third.—Tympanitic, or the drum-like percussion note, is a non-vesicular sound having the character elicited by percussing over the normal intestines; wherever heard it indicates the presence of air in conditions similar to that of the intestines, to wit.: inclosed in walls which are yielding, but neither tense nor very thick.

When elicited over the chest it may be due to the transmitted sound of the distended stomach or colon. It is obtained over the chest in pneumothorax, in moderate pleural effusions above the level of the liquid, over the seat of cavities in the pulmonary tissue, and in edema of the lungs.

The *tympanitic* percussion note differs from the normal pulmonary resonance in being more ringing in character and of a *higher pitch.*

The *amphoric* or metallic sound is in reality a concentrated tympanitic sound of high pitch, and denotes a large cavity with firm, but yet elastic, walls.

The *cracked-pot or cracked-metal* sound is another variety of the tympanitic sound. The condition most frequently producing this sound is a cavity in the lung-tissue, communicating with a bronchial tube. It requires for its development a strong, quick blow of the percussing finger, with the patient's mouth open.

Respiratory Percussion.—The percussion sound will vary greatly with the respiratory movements. If a full inspiration be taken and percussion performed, then a full expiration taken and percussion performed, and then the chest percussed during the normal respiration, slight changes in the character and pitch of the note are obtained, which otherwise would escape detection. Da Costa has designated this method, *respiratory percussion.*

Auscultatory Percussion.—This method consists in listening, with a stethoscope applied to the thorax, to the sounds elicited by percussion. "It is a serviceable means of determining with accuracy the boundaries of various organs, as those of the lungs or heart, or of the liver or spleen, and yields particularly exact results when carried out with the double stethoscope."

AUSCULTATION.

Auscultation, or listening to the sounds produced within the chest during the act of respiration, coughing, or speaking, furnishes the most reliable means of studying the condition of the lungs and heart, and is, therefore, the most valuable method of discriminating between the various conditions which may affect the lungs and heart.

Auscultation is either *immediate* or *mediate.*

It is *immediate* when the ear is applied directly to the chest, which may be either denuded or thinly covered.

It is *mediate* when the sounds are conducted to the ear by means of a tubular instrument, termed a stethoscope.

For ordinary purposes, *immediate* or direct auscultation is sufficient, but when it is desirable to analyze circumscribed sounds, as in diseases of the heart, or where the patient objects to this method, on the score of delicacy, or the auscultator objects, on account of the uncleanliness of the person examined, the stethoscope is to be preferred. Moveover, there are certain parts of the chest which can only be explored satisfactorily by the aid of a stethoscope, which instrument has the additional advantage of intensifying the sound.

In auscultation, the following rules, formulated by Da Costa, should be observed:

"1. Place yourself and your patient in a position which is the least constrained and permits of the most accurate application of the ear or stethoscope to the surface. Above all, avoid stooping, or having the head too low.

"2. Let the chest be bare, or, what is better, covered only with a towel or thin shirt.

"3. If a stethoscope be employed, apply closely to the surface, but abstain from pressing with it. This may be obviated by steadying the instrument, immediately above its expanded extremity, between the thumb and the index finger.

"4. Examine repeatedly the different portions of the chest, and compare them with one another while the patient is breathing quietly. Making him cough or draw a full breath is, at times, of service: especially the former, when he does not know how to breathe."

SOUNDS IN HEALTH.

If the ear be applied over the larynx or trachea of a healthy person, a sound is heard with both the act of inspiration and expiration. Its *intensity* is variable, its *pitch* high, and its *quality* tubular (to wit.: a current of air passing through a tube—the larynx or trachea). The duration of the sound during inspiration is somewhat longer than during expiration. A short pause follows the act of expiration.

This sound is termed the *normal laryngeal respiration,* and is identical in character, duration, and pitch with an important morbid sound, termed *bronchial respiration.*

The sound heard by placing the ear over the lung-tissue is different; it is produced in the very finest bronchial tubes and air-cells by their expansion and contraction, and is termed the *normal vesicular murmur.*

The *inspiratory portion* of the sound is of variable intensity, its pitch is low, its quality soft and breezy, designated vesicular; its duration corresponds to that of the entire act of inspiration.

The *expiratory portion* of the sound is not always perceptible; it is of feeble intensity, very low pitch, its character soft and blowing, and its duration much less than the act of inspiration.

It is to be remembered, however, that the vesicular murmur will be found to vary in the different regions on the same side, and in corresponding regions on the two sides of the chest. These variations within the range of health are especially important, and should be memorized.

Infra-clavicular Region.—The vesicular murmur in this region on either side is much more distinct than over any other part of the chest.

On the left side the *inspiratory sound* is of greater intensity, of lower pitch, and more distinctly vesicular in quality than that heard upon the right side. On the right side the *expiratory sound* is nearly or quite the same in length as the inspiratory sound, and is higher in pitch and more tubular in quality than the expiratory sound upon the left side.

Supra-scapular Region.—Owing to the small number of air-vesicles and the large number of bronchial tubes, and their close proximity to the surface, the respiratory murmur has an intense, high-pitched, tubular and expiratory quality.

Scapular Region.—Compared with the infra-clavicular region, the respiratory murmur heard over the scapula on either side is more feeble, and the vesicular quality less marked.

Inter-scapular Region.—The murmur in this region differs from the normal laryngeal breathing only in intensity and duration.

Infra-scapular Region.—The murmur in this region very closely resembles that heard in the left infra-clavicular region.

Mammary and Infra-mammary Regions.—The murmur in these regions differs from that heard in the infra-clavicular region, in being of less intensity.

Axillary and Infra-axillary Regions.—The respiratory sound in the axillary regions is as intense as in any portion of the chest. In the infra-axillary regions the intensity is less and the pitch lower.

VOICE IN HEALTH.

If the ear be applied over the larynx or trachea of a healthy person, and he be directed to count "twenty-one, twenty-two, twenty-three," in a uniform tone and with moderate force, there is perceived a strong resonance, with a sensation of concussion or shock, and a sense of vibration, thrill, or fremitus, the voice seeming to be concentrated and near the ear. Often the articulated words are distinctly transmitted (laryngophony).

The sounds heard are termed the *normal laryngeal resonance.*

If the ear or stethoscope be applied over the third rib anteriorly, on either side of the chest of a healthy person, and he be directed to count "twenty-one, twenty-two, twenty-three," in a uniform tone, with moderate force, a confused distant hum is perceived of variable intensity, accompanied with more or less vibration, thrill, or fremitus, most distinct in adults, but notably weaker in women than in men.

This sound is termed the *normal vocal resonance.*

If the ear or stethoscope be applied over the third rib anteriorly, of a healthy person, and he be directed to whisper, in a uniform manner, the words "twenty-one, twenty-two, twenty-three," there is heard a sound corresponding closely in character to the sound of expiration over the same region during the act of forced respiration; or, in other words, a feeble, low-pitched, blowing sound.

This sound is termed the *normal bronchial whisper,* and is produced by the movement of the air in the bronchial tubes during the act of respiration.

SOUNDS IN DISEASE.

The vesicular murmur may undergo, in disease, changes in its *intensity,* its *rhythm,* and in its *character.*

The **intensity** of the respiratory murmur may be:

1. *Exaggerated* or *increased.*
2. *Diminished* or *feeble.*
3. *Absent* or *suppressed.*

Exaggerated respiration differs from the normal vesicular respiration only in an increase in the intensity of the respiratory sounds. When general over one lung, it will usually indicate deficient action of other parts. In this manner an effusion compressing the lung, one-sided deposits, obstruction of the bronchial tubes by secretion, or inflammation of the lung-structure, necessitate a *supplementary* respiration in a healthy portion of the same lung or the lung upon the opposite side. From its resemblance to the loud, strong, quick respiration of young children, it has been termed *puerile* respiration. Exaggerated respiration is, therefore, to be regarded as indirect evidence of disease in some portion of the pulmonary tissue.

Diminished respiration, called also senile respiration, as being characteristic of old age, is characterized by diminished intensity and duration of the sound. In the large majority of instances the inspiration suffers the greatest, the expiratory sound not diminishing in the same proportion. In asthma, emphysema, diseases of the larynx and bronchial tubes, pleuritic pain, rheumatism or paralysis of the chest-walls, or in thickening of the pleural membrane, we observe superficial or diminished respiration. When one side of the chest is partially filled with fluid, we may hear a deep-seated but feeble breath sound.

Absent or suppressed respiration occurs whenever the action of the lung is suspended; this may be from external pressure, as when the lung is compressed by the presence of fluid or air in the pleural cavity, or when complete obstruction of the bronchial tubes prevents the air from either entering or escaping from the lungs.

The **rhythm** of the respiratory murmur may be:

1. *Interrupted or jerky.*
2. *The interval between inspiration and expiration prolonged.*
3. *Expiration prolonged.*

In health the inspiratory and expiratory sounds are even and continuous, with a short interval between each act; this may be altered in disease, and both sounds, especially the inspiratory, have an interrupted or jerky character, termed "cog-wheel respiration."

This **jerky breathing** is noted in some spasmodic affections of the air-tubes, in hysteria, the earliest stages of pleurisy, pleurodynia, and the early stages of pulmonary phthisis. It is most frequently associated with phthisis, due probably to the adhering to the walls of the finer bronchial tubes of tough mucus, which obstructs the free entrance and exit of the air; it is usually most notable under the clavicles.

The **interval between inspiration and expiration may be prolonged,** instead of these two sounds closely succeeding each other. When this occurs the inspiratory sound may be shortened, or the expiratory sound may be delayed in its commencement. If the inspiratory sound is shortened, it is the result of consolidation of the lungs; if the expiratory sound is delayed, it is the result of lessened elasticity of the lung-structure, and is most commonly associated with emphysema.

Prolonged expiration denotes that the air is obstructed in its exit from the lungs. It may be due to diminished elasticity, the result of emphysema, or from the deposit of tubercles, which impair the contractile power of the lungs. If the former, it is associated with clearness on percussion; if the latter, with impaired resonance on percussion. When prolonged expiration is detected at the apex of the lung, and is associated with impairment of the normal pulmonary resonance; it is for the most part the result of a tubercular deposit.

The **quality** of the respiratory murmur may be:

1. *Harsh,* termed *vesiculo-bronchial respiration.*
2. *Bronchial.*
3. *Cavernous.*
4. *Amphoric.*

Harsh respiration, or, as it is termed by Da Costa, *vesiculo-bronchial* respiration, is that variety in which both the inspiratory and expiratory sounds have lost their natural softness. It generally indicates more or less consolidation of lung-tissue. In normal vesicular respiration the sounds produced by the air expanding the air-cells and finer bronchial tubes obscure the sound produced by the passage of air through the larger bronchial tubes, the healthy lung being an imperfect conductor of sound, so that as soon as any portion of the lung becomes consolidated the vesicular element of the respiratory sound is diminished, the bronchial element becoming prominent. Harsh respiration is, then, a union of the vesicular and bronchial sounds, being a vesicular sound mixed with some of the qualities of a bronchial sound, the expiration being prolonged and tubular in character. It is present when the bronchial mucous membrance is swollen, as in the earlier stages of bronchitis, also in the earlier stages of phthisis and pneumonia.

Bronchial respiration is characterized by an entire absence of all the vesicular quality. *Inspiration* is of high pitch and tubular in character; *expiration* is still higher in pitch, of greâter intensity, prolonged and tubular in quality; the two sounds being separated by a brief interval. The bronchial respiration encountered in disease closely resembles that heard in health over the larynx or trachea. Whenever bronchial respiration is present where, in health, the normal vesicular murmur should be heard, it indicates consolidation of the lung-structure.

Cavernous respiration is a variety of the bronchial respiration, at least so far as the quality of the sound is concerned. It is essentially a blowing sound, yet not always heard during both the acts of inspiration and expiration, being often only perceptible in the one, and in the other mixed with gurgling sounds. Its pitch is lower than that of ordinary bronchial respiration, and its character is hollow. For its production there must be a cavity of considerable size in the lung-substance, not filled with fluid, near the surface of the chest-walls, communicating with a bronchial tube. It is met with most commonly in the last stages of pulmonary consumption, although hollow spaces of any kind, from abscess or dilatation of the bronchial tubes, occasion it.

Amphoric respiration is a blowing respiration, having a musical or metallic quality. It is a variety of bronchial respiration produced in a large cavity with firm walls, permitting the reflection of the sound. An intimation of this sound, though only an imperfect one, is produced by blowing over the mouth of an empty bottle. The amphoric character is present with both the acts of inspiration and expiration. Amphoric or metallic respiration is indicative of a large cavity, not common in phthisis, but more often heard at the upper part of a lung compressed by fluid air, as in pneumo-hydrothorax.

RÂLES.

Râles, or, as they are termed, *adventitious sounds,* because they have no analogue in the healthy state, cannot be considered as modifications of the normal respiration.

Grouped according to the anatomic situation in which they are produced, we have:

1. *Laryngeal and tracheal râles.*
2. *Bronchial râles.*
3. *Vesicular râles.*
4. *Cavernous râles.*
5. *Pleural râles.*

Râles may be divided into two groups, according to their character, to wit.: *dry* and *moist,* and may be audible either during the act of inspiration or expiration, or during both.

Dry râles, for the most part, are produced by the vibration of thick fluids which the air cannot break up, and which, therefore, temporarily lessen the caliber of the bronchial tubes. When this narrowing exists in the smaller bronchial tubes the resulting sound is high-pitched or the râle is said to be *sibilant* or whistling; when the narrowing exists in the larger bronchial tubes, the râle is low-pitched, more musical in character, or *sonorous.*

Dry râles are particularly prone to be dislodged by coughing, and when they are uninfluenced by the acts of breathing and coughing, they do not depend upon the presence of secretions, but upon the narrowing of the air-tubes from the pressure of tumors, or from a thickened fold of mucous membrane, or from a spasmodic contraction of the air-tubes.

Moist râles are those produced by the air passing through thin fluids, such as mucus, blood, serum, or pus, during the respiratory movements. When the fluid exists in the smaller bronchial tubes, the râles are termed *small bubbling, mucous,* or *subcrepitant.* When the fluid is in the large bronchial tubes, the râles are said to be *large bubbling* or mucous.

Moist râles are not persistent, but vary in intensity, and shift their position as the air drives the liquid which occasions them before it, during violent attacks of coughing, or after copious expectoration.

Laryngeal and tracheal râles are those produced within the larynx or trachea, and may be either moist or dry. The moist or bubbling sounds, produced when mucus or other liquids accumulate in this part of the air-tubes, frequently occur in the moribund state, and are then known as the "death rattles." When not due to this condition they denote either insensibility to the presence of liquid, as in stupor or coma, or inability to remove liquid by the act of expectoration, as in croup or inflammation of these parts in the very feeble.

The *dry râles* produced within the larynx or trachea are generally caused by spasm of the glottis—to wit.: laryngismus stridulus, whooping cough, croup, or from the presence of a foreign body in the part.

Bronchial râles, resulting from the passage of air through the thin liquid, occasion bubbling sounds. When the liquid is present in the large-sized bronchial tubes, the râles are said to be *large bubbling,* or large mucous râles, occurring in acute or chronic bronchitis. When the liquid is in the smaller bronchial tubes, the resulting râle is called *small bubbling,* small mucous, or *subcrepitant,* also occurring in acute or chronic bronchitis.

Bronchial râles, due to the narrowing of the tube by its spasmodic contraction, or to the presence of tough, tenacious mucus, which is put into vibration by the passage of air through the bronchial tubes, are termed *dry bronchial râles.* Frequently they are suggestive of certain familiar sounds, such as snoring, cooing, humming, or wheezing, or they are often musical tones. When produced in the smaller bronchial tubes, they are termed *sibilant,* or high-pitched râles; when produced in the larger bronchial tubes, they are termed *sonorous* or low-pitched râles. They principally occur in the dry stage of bronchitis, or during an asthmatic paroxysm.

The vesicular râle, or, as it is more commonly termed, the *crepitant râle,* is produced within the air-vesicles or at the terminal portion of the smaller bronchial tubes. It is to be distinguished from very fine bubbling sounds, or the sub-crepitant râle. *"It is a very fine sound, or rather series of very fine uniform sounds, occurring in puffs and limited to inspiration."* (Da Costa). It resembles the noise occasioned by throwing salt on the fire, or alternately pressing and separating the thumb and finger, moistened with a solution of gum arabic, and held near the ear, or rubbing together a lock of dry hair near the ear.

The *crepitant râle* is produced by the movement of fluid in the air-cells or in the finest extremities of the bronchial tubes, or by the forcing open, during the act of inspiration, of the air-cells agglutinated by exuded lymph. These sounds may be defined as being very fine, dry, crackling sounds, heard at the *end* of inspiration. They are usually present in the first stages of pneumonia, but when limited to the apices are significant of the incipient stage of phthisis.

Cavernous râles, or, as they are commonly termed, gurgling râles, are produced in a pulmonary cavity of considerable size. containing a large amount of liquid communicating freely with a bronchial tube. The sound is occasioned by the agitation of the liquid within the cavity, and may be compared to the sound produced by the boiling of liquid in a flask or large test-tube. The sound is sometimes high-pitched or musical, whence it has been termed "amphoric gurgling," but it is generally low in pitch. The râle is heard almost exclusively during the act of inspiration, and its diagnostic importance relates to the advanced stage of phthisis.

Pleural râles may be either dry or moist.

Dry pleural râles, or, as they are more commonly termed, *friction sounds,* are occasioned when the surfaces of the pleura are covered with a glutinous substance preventing the unobstructed movements of the pleural surfaces upon each other during the respiratory acts, for in health these movements occasion no sound whatever. The sounds are generally interrupted or irregular, occurring during the act of inspiration or expiration, or during both acts. The character of the sound is variable, being termed rubbing, grazing, rasping, grating, or creaking, according to the intensity of the respiratory acts and the amount of exudation.

They are distinguished by the apparent nearness of the sound to the ear, and are usually intensified by firm pressure of the stethoscope upon the chest. When the chest is fixed, especially at the lower two-thirds, and the ear applied over the seat of the sound, it will be found to have disappeared. The sound is diagnostic of the first stage of pleurisy or the pre-adhesive stage of tuberculosis of the pleura.

Moist friction sounds are produced in the same manner as those just mentioned, the exudation being softened in character. This sound is frequently confounded with moist bronchial râles, and its discrimination is often only positive by careful study of the symptoms and concomitant signs present.

Metallic tinkling is a sign of pneumo-hydrothorax with perforation of the lung, and when found, is usually diagnostic of this affection, although it occurs rarely in cases of phthisis with a large cavity, the physical conditions for its production being similar to those in pneumo-hydrothorax—to wit.: a space of considerable size containing air and liquid, the space communicating with the bronchial tubes.

It consists of a series of *tinkling sounds,* of high pitch, silvery or metallic in tone, and is very well imitated by dropping a small marble into a metallic vase. It occurs irregularly, not being present with every act of breathing, and may be produced by forced, when not heard during tranquil breathing.

Were it not for the location and the absence of concomitant signs, it might be confounded with tinkling sounds sometimes produced within the stomach and transverse colon; these latter sounds must be kept in mind in ausculting the lower chest area.

THE VOICE IN DISEASE.

The normal vocal resonance, as heard over the third rib of the chest anteriorly on each side, may have its intensity—

1. *Diminished or absent.*
2. *Increased or exaggerated.*

Or its resonance may be of the character of—

3. *Bronchophony.*
4. *Pectoriloquy.*
5. *Egophony.*
6. *Amphoric voice.*

The vocal resonance may be diminished or feeble in bronchitis with free secretion, pleurisy with effusion, or in complete consolidation of the lung-structure and the bronchial tubes.

The vocal resonance is absent in pneumothorax and in pleurisy with effusion.

Exaggerated vocal resonance differs from the normal vocal resonance in a slight increase of its density. It denotes a slight degree of solidification of lung-tissue, and is chiefly of value in the diagnosis of tuberculosis.

Bronchophony, or the voice concentrated near the ear, raised in pitch and in intensity, denotes complete consolidation of the pulmonary tissue in those parts in which the sound is abnormally present.

Pectoriloquy is complete transmission of the voice to the ear, the articulated words being distinctly recognized. It has a close resemblance to the resonance heard over the larynx in health. Its presence indicates either a pulmonary cavity or more complete consolidation—in other words, an exaggerated bronchophony.

Egophony is a modification of bronchophony, consisting in tremulousness of the voice, its character nasal or bleating, somewhat suggestive of the cry of a goat. When heard it may be considered a sign of pleurisy with slight effusion, or pleuro-pneumonia.

Amphoric voice, or "the echo," as it is sometimes called, is a musical sound, of a somewhat hollow, metallic character, like that produced by blowing into an empty bottle. It is sometimes produced in large cavities within the lung, but is especially incident to pneumo-thorax.

Increased bronchial whisper is a sound in which the whispered words are abnormally intense, and higher in pitch than the normal bronchial whisper. It has the same significance as exaggerated vocal resonance.

SUCCUSSION.

The succussion or splashing sound is pathognomonic of one affection—namely, pneumo-hydrothorax.

It is obtained by jerking the body of the patient with a quick, somewhat forcible movement, the ear being very near or in contact with the chest.

The sound is like that produced when a small keg, partially filled with liquid is shaken. The only liability to error is in confounding this splashing sound with that sometimes produced within the stomach; but attention to concomitant signs and the symptoms will always protect against this error.

ASSOCIATION OF THE PHYSICAL SIGNS

(DA COSTA).

"As many of the signs elicited by the various methods of physical diagnosis depend on the same physical conditions, they may be studied in groups. The following will be usually found to be associated.

PERCUSSION.	AUSCULATION OF RESPIRATION.	AUSCULATION. OF VOICE.	VOCAL FREMITUS.	PHYSICAL CONDITIONS.
Clear . . .	Vesicular murmur or its modification.	Normal vocal resonance.	Unimpaired.	Lung-tissue healthy or nearly so; at any rate, no increased density from deposits, etc.
Dull	Bronchial, or harsh respiration.	Broncho-phony.	Increased.	Solidification of pulmonary structure.
	Absent respiration.	Absent voice.	Diminished or absent.	Effusion into pleural sac.
Tympanitic.	Cavernous or feeble, according to cause.	Uncertain: cavernous or diminished.	Uncertain; mostly diminished.	Increased quantity of air within the chest, due to a cavity or to overdistention of the air-cells.
Amphoric or metallic.	Amphoric or metallic.	Amphoric or metallic.	Mostly diminished.	Large cavity with elastic walls.
Cracked metal sound.	Cavernous respiration.	Cavernous respiration.	Uncertain.	Generally a cavity communicating with a bronchial tube.

GENERAL SYMPTOMATOLOGY.

Dyspnea is the term used to denote difficult or impaired breathing. It may be inspiratory, expiratory, or both and there may be an increase in frequency, or depth, or both, of the respirations. It is attended by varying degrees of distress and when its severity requires the patient to sit up constantly it is called *orthopnea*.

Dyspnea may be due to obstruction of the air-passages, pressure upon the respiratory system from without by tumors, and distentention of abdomen, diseases of the lungs and pleura, heart-disease, asthma, anemia, or paralysis of muscles of respiration as the result of hemorrhage, tumors, or degeneration of the respiratory center in the medulla or toxic agents in the blood.

It may be *inspiratory* when it results from obstruction as in foreign bodies in the larynx or trachea, or it may be *expiratory* as in emphysema. A combination is the more frequent condition.

In all forms of dyspnea it is important to determine whether the shortness of breath bears any relation to exertion. Dyspnea *independent of exertion* is a serious condition and is symptomatic of severe cardiac and pulmonary disease. Dyspnea *dependent upon exertion* is less serious and is observed in health, simple debility, anemia, obesity, and somewhat moderate cardiac debility.

The *rate of respiration* varies greatly in dyspnea. Normally the respiratory rate is about 18 per minute in adult males, being somewhat more rapid in women and children. Dyspnea with slow or normal breathing is observed in diabetic coma and again the breathing may be slow and stertorous as in coma of central origin. Rapid respiration occurs in inflammatory pulmonary disease, pleurisy, painful affections of the chest muscles, heart-disease, fever, hysteria, toxic conditions affecting the respiratory centers, anemia, and morbid conditions at the base of the brain. Irregularity in the respiratory rate in dyspnea may also be observed. *Cheyne-Stokes breathing* is the term applied to this condition when the respirations gradually increase in rapidity and depth until a climax is reached after which there is a period of *apnea,* or absence of breathing. The paroxysm is then repeated. It is a serious indication and may be observed in meningitis, apoplexy, cerebral tumor, fatty degeneration of the heart, uremia, and similar conditions.

Dyspnea may be constant or paroxysmal. Constant dyspnea is always due to a persistence of its cause. Paroxysmal dyspnea is seen in asthma and cardiac affections. It may follow exertion and various central or reflex conditions. It is most marked at night.

Cough may be brought about by reflex irritation, hysteria, or direct irritation as the result of the inhalation of irritant vapors or dust, or the presence of foreign bodies but is usually due to inflammatory conditions of the pharynx, larynx, trachea, bronchi, or lungs. In the early stages of inflammation of any portion of the respiratory tract, and when excited reflexly it occurs without expectoration and is termed *dry cough.* With the occurrence of exudation and outpouring of serum, blood, etc., the cough is attended by expectoration and is called *moist cough.* Laryngeal conditions, whooping cough, hysteria, and recurrent laryngeal nerve irritation are attended by a cough having a metallic ringing intonation. This is *laryngeal* or *croupy cough.*

Sputum may vary in its several characteristics according to the morbid condition present. *Mucoid sputum* is glairy, clear, and tough and contains considerable mucin. It may be observed in health but occurs with great frequency in the early stages of acute bronchitis, pneumonia, and phthisis, in asthma, and in pulmonary edema. *Watery or serous sputum* occurs in pulmonary edema and is frothy in character. *Muco-purulent sputum* is made up of varying proportions of mucus and pus. It is encountered usually in subacute and chronic bronchitis, pneumonia in the stage of resolution, and phthisis. *Purulent sputum* is that which is made up almost entirely of pus. This is a rare condition and occurs in abscess of the lung, or adjacent viscera discharging into a bronchus, tubercular cavities, bronchiectasis, and empyema. *Nummular sputum* is that variety which occurs in round, flat disks which sink when placed in water; when spherical it is termed *globular sputum*. It accompanies advanced tuberculosis and bronchiectasis. *Fetid sputum* is that which when allowed to rest undisturbed separates into three distinct layers; the upper layer being composed of a frothy, watery material; the middle layer being made up of a greenish mucoid substance; and the bottom layer consisting of pus and débris. It is a symptom of gangrene of the lung, bronchiectasis, and advanced cavity formation. *Fibrous sputum* contains many fibrous shreds and may be seen in the various inflammations attended with fibrin formation as fibrinous bronchitis, diphtheria, etc. The *rusty sputum* is a form encountered in croupus pneumonia of which it is characteristic. It is due to the admixture of a small quantity of bright fresh blood with the thick tenacious mucus present. When the blood is retained in the vesicles and bronchioles it becomes altered and forms the *prune-juice sputum*. In gangrene, cancer, and low forms of croupous pneumonia it may be observed. A further degeneration of the blood in the lungs as in malignant disease gives rise to the production of *currant-jelly sputum*. The sputum observed in hepatic abscess contains blood, pus, bile elements, and amebæ and is called *reddish-brown sputum*.

The Microscopic Examination of the Sputum.—Blood corpuscles and alveolar cells are present in the sputum and may be detected by the aid of the microscope with or without staining.

Elastic fibers in the sputum are of great importance, as their presence signifies destruction of tissue somewhere in the respiratory tract. They are found in phthisis, gangrene, and bronchiectasis. They are usually detected in the sediment that is formed after boiling equal parts of the sputum with a ten percent solution of caustic potash. The elastic tissue remains intact in the sediment. Resort to the microscope will enable the examiner to detect the individual fibers.

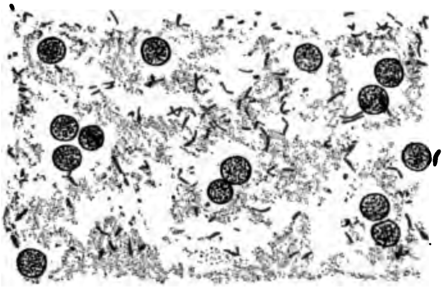

FIG. 27.—BACCILLUS TUBERCULOSIS IN SPUTUM. (*von Jaksch.*)

Connective-tissue and cartilage may in very rare instances be present in the sputum and are of grave significance. The former may accompany pulmonary abscess or gangrene, while the latter attends laryngeal ulceration.

Mucin spirals (Curschmann's spirals) are found in the sputum in bronchial asthma and occasionally in pneumonia, capillary bronchitis, and chronic pulmonary tuberculosis. They are made up of spirally arranged mucin, more or less twisted, together with epithelium and Charcot-Leyden crystals, and represent molds of the finer bronchioles. In section, they stain blue with Weigert's fibrin method.

Crystals: *Charcot-Leyden crystals* are colorless, octahedral, sharply pointed crystals resembling grains of sand. They are soluble in warm water, alkalies, acetic acid, and the mineral acids. They are particularly abundant in bronchial asthma, but may at times be detected in the sputum of acute and chronic bronchitis and tuberculosis, in leukemic blood, in semen, and in the feces. *Cholesterin crystals* occur in the sputum in tuberculosis, abscess of the lung, and liver abscess, discharging through a bronchus. They appear as thin rhombic plates, with irregular corners. *Crystals of the fatty acids,* particularly of margaric acid, are found in purulent pulmonary conditions such as gangrene, bronchiectasis, etc. They appear as long, thin, needles, occurring singly or in bundles, and not unlike elastic fibers. *Hematoid crystals* appear under the microscope as small rhomboid prisms or needles, or as free pigment particles of a brownish-yellow or ruby-red color. They occur in the sputum in pulmonary hemorrhage, abscess, cancer, gangrene, and tuberculosis.

Method for the Detection of Tubercle Bacilli.—The significance of the presence of tubercle bacilli in the sputum is undoubted—they indicate the presence of tuberculosis, but their absence does not necessarily imply an absence of the disease. The bacillus is a straight or slightly curved, non-motile organism, varying in length from two to five microns. Staining is necessary for its detection. When stained, it often presents a beaded appearance, due to the spores.(?) To examine the sputum for tubercle bacilli a small caseous particle should be selected and spread out in a very thin layer on a cover-glass or slide. It is allowed to dry in the air or by passing it through the flame of a Bunsen burner (smeared side up) three times. Ziehl's carbol-fuchsin stain (fuchsin 1; alcohol 10; five percent aqueous solution of carbolic acid 90) is then poured generously over the entire specimen, which should then be held a short distance above the flame for a few seconds until steam is formed. The slide or cover-glass should then be thoroughly washed in running water to remove the excess of the stain. Gabbet's solution (Methylene-blue 2; sulphuric acid 25; water 75) is then employed to counterstain the preparation, for which a period of about thirty seconds is required. The excess of this stain is also removed by running water, after which the specimen is dried and mounted in Canada balsam. When viewed through a 1-12 oil immersion lens, the tubercle bacilli appear as red rods on a blue background.

DISEASES OF THE NASAL PASSAGES.

ACUTE NASAL CATARRH.

Synonyms.—Acute rhinitis; acute coryza; "cold in the head."

Definition.—An acute catarrhal inflammation of the mucous membrane (pituitary or Schneiderian membrane) lining the nose and the cavities communicating with it; characterized by feverishness, feeling of fullness and discomfort in the head, and attended with discharges of fluid, watery, mucous, or muco-purulent in character.

Causes.—Atmospheric changes, exposure of the neck to a draught of cold air, or of the ankles to cold and dampness, changing from a warm to a cold atmosphere suddenly, inhalation of irritant gases and vapors, dust, and powders, such as ipecac and tobacco are the most common causes. The scrofulous taint and the rheumatic diathesis seem to render the mucous membrane susceptible to frequent attacks. Acute coryza is often present in the initial stage of the infectious fevers, such as measles, influenza, and erysipelas. Syphilis, and potassium iodid in large doses may at times produce it. Occasionally the affection seems to be contagious in character and epidemics are observed.

Pathology.—In the early stage there is hyperemia of the mucous membrane, attended with redness, swelling, and deficient secretion. This tumefaction is partly increased by an edematous infiltration, causing a quantity of colorless, salty, and very thin liquid to flow from the nose. The secretion soon assumes the character of thick, tenacious mucus or muco-pus, due to the desquamation of the epithelium of the nasal mucous membrane, and a copious generation of young cells, the hyperemia and the swelling of the membrane diminishing. The respiratory portions of the nasal fossæ are more markedly affected than are the olfactory. Rarely, and then in new-born infants and those affected with the eruptive fevers, the exudation in the nasal passages is of a fibrinous nature, somewhat similar to that observed in diphtheria.

Symptoms.—"A cold in the head" is usually preceded by a feeling of lassitude or weariness and more or less frontal headache; then occur irregular chilly sensations in the back, followed by more or less feverishness and an uncomfortable feeling of dryness in the nares, with a strong inclination to sneeze. This is soon followed by an abundant watery and saline discharge, which is continually dripping from the nostrils, or occasions an attack of sneezing followed by blowing the nose, which relieves the congested and swollen membrane for a few moments. The relief is temporary, however, the fullness of the head and difficult obstructed nasal respiration rapidly returning. The anterior nares are red and inflamed, and the eyes red and suffused with tears, through partial or entire closure of the tear-ducts. The discharge soon assumes a purulent character. The voice has a peculiar tone, rather nasal and muffled in character. Within a few days the swelling subsides and secretion lessens, health being restored in about ten days from the beginning of the attack. When the attack has almost terminated, hard crusts may form within the nostrils, either on the septum or turbinated bones, which are with difficulty expelled by blowing the nose.

Complications.—Repeated blowing of the nose and constant irritation by the discharges often causes swelling of the upper lip. The catarrhal inflammation may extend to the ethmoid or sphenoid cavities, or the frontal sinus, causing increased and severe frontal headache, or to the antrum of Highmore, causing tenderness over one or both cheeks. Extension to the eustachian tube and middle ear will cause temporary deafness and extension to the pharynx or larynx will give rise to cough. Conjunctivitis may also occur as the result of extension through the nasal duct.

Prognosis.—If the appropriate treatment is instituted promptly, mild cases will terminate favorably in about a week, and severe cases in two or more weeks. Neglected cases tend to become chronic. In very young infants, if the catarrh is not rapidly relieved, loss of flesh and strength will follow on account of the inability to nurse.

Treatment.—When due to atmospheric causes the early administration of quinin sulphate, gr. x-xv (0.6-1 Gm.), with morphin sulphate, gr. ¼ (0.016 Gm.), or Dover's powder, gr. v (0.3 Gm.), repeated in two hours, will often serve to abort an attack. A hot foot-bath or full bath, together with rest and purgation by saline laxatives, aids this abortive treatment materially. Buckley recommends the administration of bicarbonate of soda, gr. xx (1.3 Gm.) in two fluid ounces (60 Cc.) of hot water every half hour for three doses (the fourth dose to be given after an hour's interval), as an efficient abortive treatment. The following errhine used at the very onset has often proved successful:

℞.	Alumenis		
	Bismuthi subcarb.		
	Pulv. camphoræ	āā gr. xx	āā 1.3 Gm.
	Morphinæ hydrochlor. ..	gr. ij	.13 Gm.

M. Ft. chart. No. xx.

S.—Insufflate one powder in each nostril after clearing the nose.

If the attack has already developed, relief is soon afforded by the use of tincture of belladonna, ♏ii (0.12 Cc.), every hour until six doses are taken, after which one drop every two or three hours until the physiologic effects of the drug are manifest. If much fever is present tincture of aconite, ♏i-ii (0.06-0.12 Cc.), may be added. Camphor in full doses at the onset is also of value. Sajous recommends the following:

℞.	Ammonii chlor.	℈ ij	2.6 Gm.
	Tinct. opii	♏xxx	2. Cc.
	Sacch. alb.	ʒ j	4. Gm.
	Aq. camphoræ	f℥ j	30. Cc.

M. S.—One teaspoonful in water every hour or two.

Attacks of acute rhinitis, unattended by febrile reaction, may generally be lessened or promptly aborted by spraying the nares with a four percent solution of cocain hydrochlorate or adrenalin chlorid (1 to 5000). The danger of cocain habit should always be kept in mind when employing the former. With any of the previously mentioned methods of treatment may be combined one of the following errhines:

℞.	Bismuth. subnit.	ʒ vj	24. Gm.
	Pulv. acaciæ	ʒ ij	8. Gm.
	Morphinæ hydrochlor. ..	gr. ij	.13 Gm.

M. S.—Every hour or two. (Ferrier.)

Or—

℞.	Pulv. cubebæ	ʒ j	4. Gm.
	Bismuth. subnit.	ʒ ij	8. Gm.
	Morphinæ hydrochlor. ..	gr. ij	.13 Gm.

M. S.—Used by insufflation every two or three hours.

Acute coryza in nursing infants may be controlled by the insufflation into the nose of finely powdered white sugar, or equal parts of powdered white sugar and powdered camphor, or powdered sugar, ʒiv (15 Gm.), powdered camphor, ʒiv (15 Gm.), and tannic acid, gr. xl (2.6 Gm.).

Most cases of coryza in very young children are usually of syphilitic origin and require specific treatment. Symptomatic coryza necessitates no special treatment. In all cases cleansing of the nose with alkaline solutions, especially Dobell's solution, will be of value.

CHRONIC NASAL CATARRH.

Synonyms.—Chronic rhinitis; chronic coryza.

Definition.—A chronic inflammation of the mucous membrane lining the nasal passages, with more or less alteration of structure; characterized by a sensation of fullness in the nares, increased secretion, and a perversion of the senses of smell and hearing.

Causes.—It may occur as the result of repeated attacks of the acute variety; inhalation of irritating vapors and dust; syphilis and scrofula.

Pathologic Anatomy.—The mucous membrane of the nares is thickened, of a dark-red, sometimes grayish color, the superficial veins dilated and varicose, often forming polypoid enlargements (hypertrophic rhinitis). In many cases there is ulceration of the structure, with more or less loss of substance; the secretion is thick, tough, of a greenish character, and often very fetid; large collections of dried mucus are often formed upon the turbinated bones and septum (atrophic rhinitis).

Symptoms.—There is a feeling of fullness in the nose, with increased secretion of thick and greenish muco-purulent material, which, dropping posteriorly into the pharynx, causes paroxysms of "hawking," most marked in the morning immediately after rising. The sense of smell is more or less impaired and in many instances entirely abolished; hearing is diminished in many instances due to extension of the inflammation to the eustachian tube. The voice has a peculiar nasal intonation. Mouth-breathing is common on account of the nasal obstruction. There is an almost constant dull frontal headache, associated with a feeling of weight, indicating extension of the disease to the infundibulum and frontal sinus. When the affection extends to the nasal duct, lacrimation and congestion of the conjunctiva result.

In the atrophic form, there is marked shrinkage of the mucous membrane, which is pale and dry. The secretion is thick and greenish and dries within the nasal chambers, forming large, offensive crusts, the odor of which is characteristic. Ulceration is not uncommon and necrosis of the bones may occur. This form of the affection is termed *ozena*.

In all varieties, sudden changes in the atmosphere are liable to give rise to acute exacerbations, which invariably lead to exaggeration of all the symptoms.

Diagnosis.—While the symptoms are suggestive of the varieties of this affection, the diagnosis can only be made positively by rhinoscopic examination.

Prognosis.—Permanent cure is seldom obtained; the disease being so decidedly chronic and obstinate, the treatment is of necessity protracted, and the majority of patients tire of it before a complete cure is effected. In ozena, the prognosis as to cure is unfavorable, but much can be done to relieve the symptoms by appropriate treatment. Unfortunately, by reason of impairment of the sense of smell, the patient is unable to detect the offensive odor the crusts produce, and neglects treatment.

Treatment.—In the presence of evidences of syphilis, tuberculosis, rheumatism, etc., constitutional treatment should be prescribed in addition to local measures. In all cases, the general health should receive any necessary treatment.

Cleanliness of the nasal passages is of the unmost importance, and is best effected by the post-nasal syringe, with either simple or medicated tepid waters, or a cleansing solution, such as Dobell's:

℞.	Acidi carbolici	gr. j	.065 Gm.
	Sodii bicarbonat.,		
	Soddi borat.	āā gr. v	āā .3 Gm.
	Glycerini	fʒ j	4. Cc.
	Aquæ	f℥ j	30. Cc.

M. S. Use as a spray or with a proper syringe.

Or, the following combination of Sajous:

℞.	Sodii bicarb.,		
	Sodii bibor.	āā gr. viij	āā .52 Gm.
	Ext. pinus canad. fld.	♏ xv	1. Cc.
	Glycerini	fʒ ij	8. Cc.
	Aquæ	ad f℥ iv	ad 120. Cc.

M. S.— Apply with atomizer three or four times daily.

After which, decided benefit follows the use of one of the following:

℞.	Acidi borici	gr. xxx	2. Gm.
	Bismuth. subnit.	ʒ ij	8. Gm.
	Morphinæ hydrochlor.	gr. j	.065 Gm.

M.

Or—

℞.	Pulv. sanguinariæ	ʒ j	4. Gm.
	Acid. tannici	gr. v	.3 Gm.
	Pulv. camphoræ	ʒ j	4. Gm.
	Bismuth. subnit.	ʒ ij	8. Gm.

M. S.—To be used by insufflation or as a snuff every three or four hours.

Or—

℞.	Ammonii chloridi	ʒ j	4. Gm.
	Glycerini	fʒ ij	8. Cc.
	Ext. pinus canad. fld.	f℥ j	30. Cc.
	Aquæ destil.	ad f℥ ij	ad 60. Cc.

M. S.—Five or ten drops, dropped into each nostril two or three times a day, or applied with a camel's-hair brush.

Or the following pleasant mixture may be applied to each nostril:

℞.	Tinct. benzoin.	fʒ iv	15. Cc.
	Tinct. guaiaci	fʒ j	4. Cc.
	Chloroformi	♏x	.6 Cc.
	Tinct. myrrh.	fʒ ss	2. Cc.
	Ol. amygd.	♏v	.3 Cc.

M. S.—A few drops in each nostril once a day.

Frequently the mucous membrane becomes greatly hypertrophied and requires the use of the galvanocautery or caustics to remove the obstruction. Polyps may also form and should be removed by the snare. In atrophic rhinitis or ozena difficulty is often encountered in removing the crusts before making any local application. This may be overcome by the use of peroxid of hydrogen or ordinary coal-oil and generous douching with an alkaline solution. Oily sprays, such as liquid vaselin, or albolene, with or without the addition of menthol, eucalyptol, or thymol, may then be employed. Boroglycerin is also of great value.

DISEASES OF THE LARYNX.

ACUTE CATARRHAL LARYNGITIS.

Synonyms.—Catarrhal laryngitis; "sore throat."

Definition.—An acute catarrhal inflammation of the mucous membrane of the larynx; characterized by feverishness, diminished or suppressed voice, painful deglutition, and more or less difficulty of respiration.

Causes.—Atmospheric changes, cold draughts of air directly inhaled, or undue exposure of any or all parts of the body to the same, cold and wet feet, inhalation of dust or irritating vapors, such as gases, smoke, ammonia, etc., prolonged efforts at singing or speaking in public, especially when under difficulties, impacted foreign body, and infectious fevers are the most common causes. In children it may be due to violent fits of crying.

Pathologic Anatomy.—The mucous membrane is congested and swollen, and the secretion greatly diminished. In many cases only portions of the laryngeal mucous membrane are involved. The inflamed membrane returns to its normal condition very shortly, and the secretion is then increased.

Symptoms.—The attack begins rather suddenly with a feeling of dryness, rawness, and tickling, referred to the larynx with the sensation of the presence of a foreign body in the throat, and with hoarseness and a disposition to cough. Deglutition causes pain by the upward movement of the larynx and by the pressure of the food on the larynx as it passes along the gullet. Attempts at speaking are attended with more or less distress and the larynx is tender on pressure. Coughing, of a noisy, harsh, hoarse, or toneless character is present from the onset, and is attended by a sensation of scratching in the larynx. The first day or two there is scanty expectoration, but in a short time the secretion is increased, giving the cough a loose character. In the early stages the sputa may be slightly streaked with blood. Rarely a hemorrhage occurs from the mucous membrane of the larynx. The voice is at first decidedly hoarse, soon followed by complete aphonia. The respiration is but slightly, if at all, affected in adults, except when there is marked edema. There may be more or less febrile reaction. In children the onset is attended with fever, white coated tongue, frequent, tense pulse, hot skin and flushed face, embarrassed respiration, the voice hoarse and whispering, with harsh, ringing, croupy cough and great restlessness. During the night the child is subject to suffocative attacks (laryngismus stridulus). Similar paroxysms may also occur in highly sensitive adults.

Laryngoscopic Appearances.—These vary with the severity of the attack and the stage of the inspection. In *mild cases,* at an early period, the mucous membrane presents a bright red appearance. *Severe cases* present, in addition to the bright redness, swelling of the mucous membrane to such an extent at times as to conceal the vocal cords, they appearing only as slender threads of a reddish tint. At times the mucous membrane presents the appearance of erosions or ulcerations, due to the desquamation of the epithelium.

Prognosis.—Simple catarrhal laryngitis never terminates fatally and runs its course usually in one week, but may be prolonged for two or three weeks in severe cases. When edema is present in addition, there is always danger from asphyxia.

Treatment.—The patient should be confined to an apartment of uniform temperature, the air being kept moist by the vapor of boiling water. Attempts to use the voice should be discountenanced. At the very beginning of an attack the feet should be placed in a hot mustard foot-bath and either a saline or mercurial purgative should be administered. Prompt action of the skin at this stage will frequently aid in shortening the attack. For this purpose Dover's powder, gr. iii (0.2 Gm.), combined with potassium nitrate, gr. iii (0.2 Gm.), should be administered every three or four hours. If there is much febrile reaction, benefit will be obtained from the use of tincture of aconite, ♏ i-ii (0.06-0.12 Cc.), every half hour until five doses are taken, after which it should be given every hour or two, combined with tincture of opium, ♏ i-v (0.06-0.3 Cc.). Diaphoresis is also of value and may be obtained by the administration of ammonium and potassium tartrate, gr. 1-20 to 1-30 (0.003-0.002 Gm.), every hour, or by the hypodermic injection of pilocarpin hydrochlorate, gr. ⅓ (0.022 Gm.).

For children, several doses of the following should be given a couple of hours apart, until the bowels are freely moved:

℞.	Hydrargyri chloridi mitis..	gr. ⅛	.008 Gm.
	Pulvis ipecacuanhæ	gr. ⅛	.008 Gm.
	Sacch. lactis	gr. ij	.13 Gm.

M. S.—One dose.

To be followed by—

℞.	Potassii citrat.	℈ iv	5.3 Gm.
	Tinct. aconiti	♏ v	.3 Cc.
	Tinct. opii camphorat. ..	fʒ ij-iv	8.-15. Cc.
	Syr. scillæ	fʒ ij	8. Cc.
	Syr. tolu	ad f℥ iij	ad 90. Cc.

M. S.—One teaspoonful every two hours.

If a tendency to spasm of the glottis obtains, full doses of the bromids should be administered at once.

Inhalations from the onset are not only soothing, but curative, in their actions. Either of the following are recommended:

℞.	Infusi humuli ....Oj	480. Cc.
	Vinegar ....f℥ ss-j	15.-30. Cc.

M. S.—Inhale hot every hour.

Or—

℞.	Tinct. benzoin. comp. ..fʒ j-ij	4.-8. Cc.
	Aquæ bull. ....Oj	480. Cc.

M. S.—Inhale hourly.

The local application of cocain is of great benefit. A hot pack should also be kept constantly wrapped about the throat, and if its application is preceded by the temporary use of a weak mustard plaster, the relief afforded is more rapidly obtained.

Attacks of acute laryngitis following efforts at singing or speaking in public, are wonderfully benefitted by the use of dilute nitric acid, ♏ii-v (0.12-0.3 Cc.), every hour; or atropin sulphate, gr. 1-300 (0.00022 Gm.), every hour for several doses.

The onset of edema calls for cold applications, scarification, astringent applications, and if asphyxia threatens, tracheotomy.

EDEMATOUS LARYNGITIS.

Synonym.—Edema of the glottis.

Definition.—An acute inflammation of the mucous membrane of the larynx and that about the glottis, with an infiltration of the areolar tissues by a serous, sero-purulent, or purulent fluid; characterized by obstructed or stridulous breathing and dysphonia or aphonia.

Causes.—It may occur in the course of acute laryngitis, suppuration in or about the throat or tonsils, facial erysipelas, scarlatina, small-pox, diphtheria, Bright's disease, or urticaria. Burns, scalds, swallowing of caustic substances, and ulcerative affections such as tuberculosis and syphilis may produce it. It is rare in children, most cases occurring in men between the ages of 20 and 35 years.

Pathologic Anatomy.—Infiltration into the loose connective tissue of the ary-epiglottic folds, the glosso-epiglottic ligament, the base of the epiglottis, and the inter-arytenoid space is the principal change. If the true vocal cords are inflamed, their color changes, and instead of appearing white, glistening, and brilliant, they are dull, grayish-red, or violet-red in patches. If the swelling be the result of purulent infiltration, the parts present a deeply congested color, with here and there spots of a yellowish hue. Serous infiltration, sufficient to cause fatal edema, disappears with death, leaving but slight traces to account for the formidable symptoms.

Symptoms.—The onset is much the same as a simple catarrhal laryngitis with a gradually increasing impediment to the respiration. The patient experiences the sensation of a foreign body in the throat, and after a short time difficulty of breathing, which ultimately threatens suffocation. The deglutition is rendered difficult owing to the swelling of the epiglottis; the voice, at first muffled, gradually becomes weaker and weaker, until finally it is almost extinct; the cough at first is dry and harsh, but as the infiltration increases it becomes stridulous and suppressed; there is no expectoration, except after great effort to clear the throat, when a little frothy mucus is raised. The difficulty of respiration, as the disease progresses, becomes greater and greater, and the paroxysms of impending suffocation more frequent. The inspiration is accompanied by a whistling sound characteristic of the narrow condition of the glottis; the patient sits up in bed, his mouth open, gasping for breath, his eyes protruding, the whole body trembling with intense convulsive movements, and after a time a general cyanosis commences, the face assuming a bluish hue, all these symptoms continuing for a few moments, when slight relief occurs, to be again followed by another paroxysm, in one of which, if nature or art does not afford prompt relief, death occurs from asphyxia.

A physical examination of the parts may be made by gently passing the finger into the throat, when the epiglottis may be felt very much thickened, and the ary-epiglottic folds may have attained such tumefaction as to convey to the finger an impression similar to that which is given by touching the tonsils.

Laryngoscopic Appearance.—The mucous membrane has a bright red appearance. The epiglottis has the appearance of a semi-transparent roll-like body, or it is often merely erect and tense. It is this condition of the epiglottis which explains the pain and difficulty in deglutition. Rarely the vocal cords are infiltrated.

Diagnosis.—Any disease which gives rise to dyspnea may simulate edematous laryngitis, but the history of the case, together with a laryngoscopic examination, will generally furnish conclusive evidence of the nature of the malady.

Prognosis.—The outlook is unfavorable; about one-half of the cases terminate fatally. If early and vigorous treatment be instituted recovery is possible, but without it asphyxia and death are the inevitable results. Even after the local obstruction has been removed, the patient is liable to perish subsequently from exhaustion, blood-poisoning, or pulmonic complications. The duration varies from a few hours to several days.

Treatment.—Prompt local treatment is necessary to relieve the obstruction. Leeches placed externally over the larynx may be of value in reducing the edema in mild cases. The persistent use of ice-pellets early in the attack, swallowed or held far back in the mouth until dissolved, is recommended by Niemeyer. The hypodermic injection of pilocarpin hydrochlorate, gr. ⅓ (0.022 Gm.), until free salivation and diaphoresis are produced, is of great value, care being taken to avoid cardiac depression. Mackenzie advocates keeping the patient constantly under the influence of potassium bromid. The inhalation or spray of a strong solution of tannic acid (Trousseau), or local application of Monsel's solution, full strength or diluted, (Da Costa), may also be employed. The blanching and shrinking effect of cocain and adrenalin would seem to recommend these drugs in this condition.

Relief may be afforded in the early stage by scarification of the edematous tissues, guiding the instrument by the index finger of the opposite hand. If the various measures already mentioned fail, tracheotomy is indicated; in those cases of rapid and sudden infiltration of the glottis or larynx occurring in Bright's disease, erysipelas, scarlatina, or syphilis of the larynx, *tracheotomy should be performed at once.* Intubation will also at times relieve the obstructed breathing.

In all cases, food and stimulants should be administered, preferably by the rectum, as swallowing is difficult and serves to aggravate the condition. If the infiltration becomes purulent, quinin sulphate, gr. v (0.3 Gm.), every four hours is indicated in addition.

SPASMODIC LARYNGITIS.

Synonyms.—Spasmodic croup; false croup; catarrhal croup.

Definition.—A catarrhal inflammation of the mucous membrane of the larynx, associated with temporary spasmodic contraction of the glottis; characterized by paroxysmal coughing, difficulty of breathing, and attacks of threatening suffocation.

Causes.—Atmospheric changes or "taking cold," excesses in eating and drinking, excitement, and violent emotion, are given as causes for simple croup.

Pathologic Anatomy.—Congestion of the mucous membrane of the larynx, with slight swelling and deficient secretion, are the only changes that have thus far been noted.

Symptoms.—The attack occurs chiefly during the night, the child on retiring having either its usual health, or perhaps being a little feverish. After several hours of sleep the child is suddenly awakened by a paroxysm of suffocation, and a dry, harsh, ringing cough. After half an hour or an hour or two the breathing becomes easier, and the cough less "croupy;" the skin is covered with more or less perspiration, and the child falls asleep. The next day there is present cough of a loose character, the respiration being about normal. If no treatment be instituted, the same phenomena occur on the second night, the child being apparently well during the second day, the cough being less in amount; phenomena of a similar character, but of much less severity, are present the third night, after which the disease usually disappears.

If the symptoms of the first paroxysm continue pronounced for two or three days, there is a strong probability that the inflammation may become fibrinous in character, or that true croup may develop.

Diagnosis.—The history, course, and absence of marked constitutional disturbances will distinguish this affection from *diphtheria.* In *laryngismus stridulus,* there is a history of rachitis, and an absence of catarrhal symptoms.

Prognosis.—Spasmodic or simple croup always terminates favorably.

Treatment.—During the paroxysm, the child should at once be placed in a hot bath and hot or cold compresses should be applied to the throat. These measures should be preceded or followed by the administration of a mild emetic. The syrup or wine of ipecac, in doses of ʒss-ʒi (2-4 Cc.), every few minutes until vomiting is produced, is very efficient. Bartholow recommends turpeth mineral, gr. i-iii (0.065-0.2 Gm.); Da Costa suggests the cautious use of apomorphin hydrochlorate, gr. 1-10 (0.006 Gm.), hypodermically. The late Charles D. Meigs always used powdered alum alone or with syrup of ipecac. Powdered alum is of great value in teaspoonful doses, administered in honey or molasses and repeated in fifteen minutes, until vomiting is produced. In the absence of these means of inducing emesis, irritation of the fauces by a feather or by the finger, will bring about the desired result. In very severe paroxysms the inhalation of chloroform may be necessary.

As soon as the paroxysm has been broken, a laxative should be given. Calomel, gr. ii (0.13 Gm.), and sodium bicarbonate, gr. iii (0.2 Gm.), should be administered and followed in six or eight hours by a dose of castor oil or magnesia. During the intervals between the paroxysms, small doses, ♏v-x (0.33-0.66 Cc.), of the syrup or wine of ipecac, or the following should be given:

℞. Tincturæ aconiti	♏viij	.5 Cc.
Syr. ipecacuanhæ	fʒ iss	6. Cc.
Tincturæ opii camphorat.	fʒ iij	12. Cc.
Liquor potassii citratis.	ad f℥ iij	ad 90. Cc.

M. S.—One teaspoonful every hour or two.

CROUPOUS LARYNGITIS.

Synonyms.—Membranous croup; true croup.

Definition.—An acute inflammation of the mucous membrane of the larynx, attended with the exudation of a tough secretion—the false membrane—and the occurrence of spasm of the glottis; characterized by febrile reaction, frequent ringing cough, dyspnea, with loud respiratory sound, and altered or extinct voice, showing a strong tendency toward death by asphyxia.

Causes.—It is most common in humid winters and is a disease of childhood, occurring most commonly in strong, vigorous, well-nourished males. Certain families present a strong hereditary tendency toward the disease. Most authorities believe this affection to be identical with laryngeal diphtheria. This view, however, is not held by the author.

Pathologic Anatomy.—The earliest change is intense hyperemia of the laryngeal mucous membrane with redness, swelling, and edema. A grayish pellicle soon forms which eventually becomes the opaque, false membrane, which differs in thickness and adhesiveness in different portions of the larynx. In all cases, the false membrane is present on the vocal cords and inner surface of the epiglottis. The first membrane softens by the exuded serum, and is then dislodged by coughing or vomiting, to be followed by successive deposits. When the false membrane is detached, the mucous membrane of the larynx is found unaffected, so far as the loss of structure is concerned. Several successive crops of membrane may form after the detachment, or it may entirely cease to form after the removal of the first exudation.

On *microscopic examination* the false membrane is found to be composed of a fine network of fibrille, holding in their interstices leukocytes of an albuminous or fibrinous nature.

The false membrane may extend into the pharynx, but especially is it liable to extend into the trachea and bronchial tubes, and, as the inflammation extends downward, the character of the exudation changes from fibrinous to muco-purulent.

Symptoms.—The onset of "true croup" is either suddenly, by an attack of spasmodic croup, or gradually, as an acute catarrh of the larynx, rapidly increasing in severity, with a feeling of heat in the throat, huskiness of the voice, harsh cough, fever, and thirst, the hoarseness soon becoming marked, and the cough having a metallic, "croupy" character, rapidly changing to a stridulous, husky sound; every few minutes the child takes a sudden, deep, stridulous inspiration, the voice becoming more and more husky. Difficulty of breathing now follows; the child is unable to lie down, or if exhausted by the efforts at inspiration, it is quiet for a moment, it soons starts up in fright, breathing more heavily, with a shrill, whistling inspiration. Soon, from the narrowing of the glottis due to the presence of the membrane, the expiration becomes difficult and noisy, and suffocation seems imminent from the paroxysmal attacks of spasm of the glottis; the child tosses wildly about, tears at its throat, as if to remove some obstacle, the face becoming cyanosed, the alæ of the nose working rapidly, the mouth wide open, the inspiratory efforts gasping, the body covered with a profuse sweat, and death seems imminent, when, suddenly, the spasm is relaxed, air enters the chest, the breathing becomes somewhat easier, and the child, exhausted and partially stupefied, drops into a fitful sleep of a few moments' duration.

The suffocative attacks return at short intervals, or there occur decided remissions between them, considerable portions of the false membrane being expelled, allowing the child to fall into a refreshing sleep.

In those cases which tend to a favorable termination, the appearance of improvement noted between the suffocative attacks is maintained, the paroxysms of suffocation becoming less frequent, the expectoration of membrane more marked, the difficulty of breathing lessens, the cough loosening, the voice gradually returning, and the fever, which has been more or less high during the attack, disappearing.

If, instead of improvement, the case tends toward a fatal termination, the suffocative attacks become more frequent, expectoration is absent, the voice and cough inaudible, although the efforts at speaking and coughing are visible, the difficulty of breathing continues, the respirations becoming more frequent and shallow, but without whistling and stridor, the cyanosis deepens, the countenance has an indifferent, drowsy, and stupid look, the eyes dull and nearly closed, with symptoms of depression, the pulse rapid and weak, the surface covered with a cold, clammy sweat, the extremities cold, and stupor and insensibility become more marked, the child dying of carbonic-acid-poisoning or asphyxia.

Duration.—The duration of true croup is about one week, rarely ten days.

Diagnosis.—Edema of the glottis might be mistaken for croup until the period of the formation of the characteristic membrane. The chief points of distinction from the onset are, however, absence of fever, paroxysmal attacks of difficult respiration, followed by a complete return to the normal condition. Edema of the glottis is rare in childhood.

The following are the chief points of difference between croup and laryngeal diphtheria:

CROUP.	DIPHTHERIA.
A local disease.	A constitutional disease.
Begins in trachea and extends up.	Begins at tonsils and extends down.
Exudation never cutaneous.	Exudation often cutaneous.
No pain in swallowing.	Often severe pain in swallowing.
No swelling of submaxillary and lymphatic glands.	Swelling of submaxillary and lymphatic glands.

Cough always present and often reduced to a mere whistle with peculiar metallic ring.	Seldom much cough and then only hoarse.
Not traceable to bad drainage.	Often traceable to bad drainage.
Seldom occurs in adults.	Often occurs in adults.
Neither contagious nor infectious.	Both contagious and infectious, both before and after death.
A sthenic disease.	An asthenic disease.
Membrane does not extend to nares.	Often extends to nares and many other parts.
No symptoms of septicemia.	Septicemia generally present.
No albuminuria.	Albuminuria frequent.
Neither attended with nor followed by paralysis.	Paralysis not uncommon.
Death seldom caused by syncope.	Death from syncope common.
Death due to suffocation.	Death frequently results from other causes.
Absence of a specific microörganism.	Presence of the Klebs-Loeffler bacillus.

Prognosis.—The affection is very fatal and the danger is proportionate to the age and feebleness of the patient. Intubation has been of great service in lessening its mortality.

Unfavorable symptoms are: Loud, stridulous, inspiratory and expiratory sounds, laborious and prolonged expiration, depression of the base of the thorax during inspiration, whispering voice or complete aphonia, congestion of the face and neck, stupor, weak, rapid, and irregular pulse, cold extremities, and a cold, clammy perspiration.

Favorable symptoms are: Expectoration of false membrane, decrease of the stridulous respiration, voice changing from whispering to hoarseness, looseness of the cough, moderation of the fever, and an improvement in the general condition.

Treatment.—The indications are to detach and remove the false membrane, to prevent its reformation, to prevent the attacks of spasm of the glottis, and to maintain the patient's strength.

For the first indication emetics are of great value. Barker recommends turpeth mineral, gr. ii (0.13 Gm.), for a child two years of age, repeating the dose as the obstructed breathing requires it. The unnecessary administration of emetics, however, should be avoided.

To prevent the formation of the membranous exudation, ammonium and potassium tartrate, gr. 1-30 to 1-20 (0.002-0.003 Gm.), quinin sulphate, gr. v (0.3 Gm.) every three hours until six doses have been taken, and bichlorid of mercury, gr. 1-48 to 1-20 (0.0015-0.003 Gm.) every two hours may be employed. The following formulas are also of value:

℞. Hydrargyri chloridi mitis.gr. ⅛-¼-½ .008-.016-.032 Gm
Sodii bicarbonatis ......gr. ij .13 Gm.
Pulvis ipecacuanhæ .....gr. 1-12-⅙ .005-.011 Gm.
M. S.—One powder every two hours.

℞. Antimonii sulphurati ....gr. ⅛ .008 Gm.
Pulv. ipecacuanhæ et opii.gr. ½ .032 Gm.
M. S.—In powder every two hours. (Da Costa).

Or—

℞. Hydrargyri chloridi mitis...gr. ⅛ .008 Gm.
Pulvis ipecacuanhæ et opii.gr. ½ .032 Gm.
M. S.—In powder every two hours.

To prevent the spasms of the glottis, small doses of opium, in the form of Dover's powder, or full doses of the bromids, preference being given to ammonium bromid, should be administered.

The strength of the patient should be maintained by predigested liquid food, nutrient enemas, and alcohol, strychnin, ammonium carbonate, and similar drugs. As soon as emesis has subsided intubation should be performed.

Locally, hot or cold compresses, sinapisms, or flying-blisters, may be applied to the neck to relieve the distress. Inhalations of oxygen or the vapor of slaked, freshly-burned lime, are efficient in detaching the membrane. Peroxid of hydrogen locally applied may also be of value, but caustic or irritating applications to the fauces or larynx are positively contraindicated.

Niemeyer advises, in cases showing carbonic acid poisoning from obstruction of respiration due to accumulation of membrane, pouring from a moderate height of a few gallons of cold water over the head, nape, and back of the child; the shock it produces always causes the child to revive for a while, and to cough vigorously, thus expectorating large quantities of the membrane, but this procedure will become obsolete in proportion as intubation is practised. If the exudation still continues, regardless of the means employed, the propriety of tracheotomy must be considered.

LARYNGISMUS STRIDULUS.

Synonyms.—Spasm of the glottis; pesudo-croup; Millar's asthma; thymic asthma: "Kopp's asthma"; tetany; child-crowing.

Definition.—A spasm of the muscles of the larynx innervated by the inferior or recurrent laryngeal nerves; characterized by a sudden development of dyspnea and deficient oxygenation of the blood.

Mackenzie describes it as "a form of convulsion occurring in ill-nourished infants, characterized by spasmodic action of the abductors of the vocal cords, and in severe cases by spasm of the diaphragm and intercostal muscles."

Causes.—The affection is most common in young children, as the result of teething, laryngitis, scrofula, fright, and rachitis. It occasionally occurs in adults. It may be hereditary. Many observers believe it to be a form of tetany.

Pathology.—Death rarely occurs, and in consequence the morbid anatomy is as yet undetermined. The mechanism consists in an irritation of the superior laryngeal nerve,—the afferent nerve,—whose function is to supply the mucous lining of the larynx with sensibility, whence is reflected through the inferior laryngeal nerve—the efferent nerve—the motor influence resulting in the spasm of the laryngeal muscles.

Symptoms.—The spasm of the laryngeal muscles is of sudden onset, and usually after nightfall. The child may have been in perfect health, to all appearances, on retiring, or it may have shown symptoms of catarrh of the upper air passages, or been suffering from gastrointestinal or dental irritation. The child awakens suddenly, coughing in a metallic, resonant tone—the croupy cough—and with great dyspnea, with loud, crowing, stridulous inspirations, the result of narrowing of the larynx from spasm, and with wheezy, stridulous expirations. The entrance of air is so greatly obstructed that all the accessory muscles of respiration are called into use; the lips and finger nails become blue, the surface cold, the countenance anxious, and the inferior portion of the chest is drawn in, instead of being expanded, during inspiration. General convulsions occur at times, during a paroxysm, also strabismus, and involuntary discharge of the feces and the urine.

The paroxysm continues from half an hour to an hour or more, to return after a few hours' sleep or during the following night; the cough, during the day, having the croupy character.

Diagnosis.—The non-febrile and distinctly intermittent character of the affection with its peculiar crowing inspiration differentiates it from croup and the laryngeal conditions.

Prognosis.—Favorable. Death from suffocation during the paroxysm may occur in very young and debilitated children, but it is a very rare termination.

Treatment.—The inhalation of a few drops of chloroform or amyl nitrite will serve to relieve the paroxysm. Nitroglycerin in small but frequently repeated doses or the following combination is a valuable antispasmodic:

℞.	Potassii bromidi	ʒ ij	8. Gm.
	Chloral	gr. xxxij	2. Gm.
	Syr. aurantii cort.	f℥ j	30. Cc.
	Aquæ menth.	f℥ j	30. Cc.

M. S.—One teaspoonful every half hour.

Mackenzie advises the use of musk during the attack if the child can swallow; and if not, then as soon as the child can take it, and continued at intervals for a day or two. His formula is as follows:

℞.	Moschi	gr. iss	.10 Gm.
	Sacch. alb.	gr. ij	.13 Gm.
	Pulv. acaciæ	gr. ij	.13 Gm.
	Syr. aurantii flor.		
	Aquæ	āā ad fʒ j	āā 4. Cc.

M. S.—One dose.

The high price of musk, however, prohibits its general use.

After the attacks have been suspended, the tendency to recurrence is prevented by continued use of potassium bromid in moderate doses. Cases due to indigestion are greatly relieved during the paroxysm by the administration of an emetic.

Locally, the hot pack, alternating with the cold pack, should be applied to the throat continuously. The vapor of boiling water should be inhaled in addition.

After the attack has subsided, calomel followed by magnesia or castor oil should be given. All farinaceous substances should be eliminated from the diet and tonics should be administered. The rachitic factor in the disease should also receive attention.

CHRONIC LARYNGITIS.

Causes.—Simple chronic catarrhal inflammation of the larynx may be due to repeated acute attacks or may follow a persistence of the same causes that produce the acute variety such as overuse of the voice, irritation of smoke, vapors, etc., and excessive use of alcohol and tobacco.

Pathologic Anatomy.—Redness and swelling are present and there is more or less thickening of the parts concerned in the production of the voice. Relaxation of one or both vocal cords may be observed. Superficial erosions, distention of the follicles, and villous outgrowths on the cords may be present.

Symptoms.—Hoarseness and discomfort in the use of the voice are the most prominent symptoms. Aphonia may occur. There is a great tendency to cough but expectoration is scanty and mucoid in character. Inspection reveals swelling, congestion, and a granular appearance of the larynx.

Prognosis.—Owing to the persistence of the causes and the inability of the patient to co-operate in the treatment, complete recovery is not common. Under more favorable circumstances, the prognosis is fairly good.

Treatment.—The various causes should be ascertained and promptly removed. Smoking, and drinking of alcoholic beverages should be prohibited and the patient taught to properly use the voice. Systematic exercises, fresh air, and tonic treatment are indicated. Associated nasal and pharyngeal affections should receive attention. Astringent sprays such as alum (3 percent) solution, tannin solution (1 to 2 percent), sulphate of zinc (3 percent solution), etc., are of great value and should be preceded by cleansing of the pharynx and larynx with Dobell's solution or some similar alkaline mixture. The inhalation of steam charged with some volatile substance such as benzoin, benzoic acid, or cubebs is also beneficial. Troches containing benzoic acid (½ gr.), cubebs (1 to 2 gr.), ammonium chlorid (3 to 5 gr.), potassium chlorate (5 gr.), etc., are productive of good results. Insufflation of dry powders such as starch and tannic acid (equal parts), alum and starch (equal parts), iodoform, and similar substances, into the larynx often affords relief. The direct application of silver nitrate solution (10 to 15 grains to the ounce) or a solution of resorcin (10 percent in glycerin) to the diseased areas is efficacious in many cases.

SYPHILITIC LARYNGITIS.

Syphilis of larynx may manifest itself as a diffuse non-distinctive catarrhal inflammation, moist papules or "mucous patches," or ulceration (gumma). The mucous patches may be found on the epiglottis, in the laryngeal wall, and on the epiglottidean folds but rarely on the vocal cords. They are seldom replaced by ulceration. Gumma of the larynx is followed by circular, deep, and sharply marginated ulcers. Rapid necrosis of the cartilages is common, and the resulting cicatrix may give rise to stenosis. The symptoms are hoarseness, cough, more or less loss of voice, and pain on deglutition.

Diagnosis.—The history, the rapidly spreading circumscribed ulcers, and the results of specific treatment aid greatly in distinguishing this affection from tubercular laryngitis in which there are manifestations of tuberculosis elsewhere in the body.

Prognosis.—Under proper treatment the ulcers heal rapidly, but the resulting cicatrices may permanently impair the voice.

Treatment.—Mercury and the iodids should be administered in full doses to the point of tolerance. Locally, astringent and alkaline sprays, together with applications of silver nitrate (solid stick or in solution) or chromic acid solution (12.5 percent) to the ulcers, are of great value. Insufflation of iodoform is also of value. The galvano-cautery directly applied to the ulcers is also recommended. Zinc chlorid, copper sulphate, and similar astringents may be used with benefit.

TUBERCULOUS LARYNGITIS.

Synonyms.—Laryngeal phthisis; throat consumption.

Definition.—A tuberculous inflammation of the larynx, characterized by ulceration, pain on deglutition, cough, weakness of voice, hectic fever, and progressive emaciation.

Cause.—The affection is produced by the tubercle bacillus, and may be primary, but is usually secondary to some other focus of tuberculosis, usually in the lungs.

Pathologic Anatomy.—All laryngeal affections in the course of phthisis are not necessarily tubercular. True tubercular laryngitis begins with redness of the mucous membrane, showing scattered tubercles. The tubercles show a strong tendency to cluster, then soften, leaving shallow, irregular ulcers. The ulcers are covered with a grayish exudate. The mucous tissue round about the ulcers is thickened. The ulcers may, and generally do, erode the true vocal cords, often entirely destroying them. The ulcers slowly extend in all directions, destroying the tissues attacked. The epiglottis may be entirely destroyed.

Symptoms.—The first symptom is a change in the voice—*huskiness;* this, associated with symptoms of ill-health, is always a warning to the physician. The husky voice may proceed until it is but a painful whisper. Cough of an irritating, painful character is present, associated with slight expectoration. Painful and difficult deglutition (dysphagia) is a very constant and distressing symptom. There is the remitting fever so characteristic of tuberculosis, with night-sweats, loss of appetite, loss of flesh, and insomnia.

Laryngoscopic examination reveals the characteristic broad, shallow, irregular, grayish ulcers, with the thickened surrounding mucous membrane. The vocal cords show infiltration and thickening or ulceration.

Diagnosis.—While the broad, shallow, irregular ulcers are fairly characteristic of this disease, no positive diagnosis can be made until the sputum is examined and tubercle bacilli found therein.

Prognosis.—Unfavorable.

Treatment.—The general treatment is that of tuberculosis elsewhere in the body. The diet should be liquid and concentrated, on account of the distress and difficulty in swallowing. Much can be done by local treatment to render the patient comfortable. The application of lactic acid (20, 40, or 60 percent solution) to the larynx is very beneficial. Cocain similarly employed is also of value in relieving the pain and dysphagia. Hydrogen peroxid, silver nitrate, and menthol may be used with good results. Curetting of the ulcers and applying iodoform in emulsion or with morphin sulphate has been practiced with benefit. Alkaline washes and sedative inhalations are also recommended.

DISEASES OF THE BRONCHIAL TUBES.

ACUTE BRONCHITIS.

Synonyms.—Bronchial catarrh; acute bronchial catarrh; "cold on the chest."

Definition.—An acute catarrhal inflammation of the bronchial tubes of the larger, middle, and third size; characterized by fever, substernal pain, a feeling of thoracic constriction, oppression in breathing, and at first scanty, followed by more or less profuse, expectoration.

Causes.—It is most frequent in childhood, especially during the period of dentition, when there exists a strong tendency to catarrh of the mucous membranes in general and of the bronchi in particular. In old age the predisposition again returns. Inhalation of irritants such as dust, smoke, and air, too hot or too cold, is also a common cause. The affection is more common in climates characterized by considerable moisture of the atmosphere, combined with a low temperature, and especially where there are sudden and marked variations. Chronic heart-disease, uric acid diathesis, and exposure to cold and wet are potent etiologic factors. It accompanies the infectious fevers such as typhoid fever, influenza, whooping cough, and measles. The exciting cause is a microörganism. Staphylococci, streptococci, or pneumococci may be found in the sputum.

Pathologic Anatomy.—The mucous membrane of the bronchial tubes is at first congested, swollen, and edematous. Secretion is diminished. Later, there are increased secretion and overgrowth and desquamation of the epithelium together with proliferation of young cells and leukocytic infiltration. The expectoration is then of a yellowish color (muco-purulent). In the early stage the scant expectoration may be streaked with blood due to rupture of the distended capillaries.

In cases of bronchitis following the exanthemata, or in scrofulous patients, the bronchial glands participate in the inflammation, becoming hyperemic, swollen, and filled with secretion, and not infrequently the glandular elements undergo a hyperplasia, and finally the "cheesy" degeneration.

Symptoms.—The invasion may be attended by nasal or laryngeal catarrh, or both. Usually the onset begins with chilliness, followed by flushes of heat, aching pain of a contused character in the limbs, joints, and trunk with a sense of fatigue and loss of energy, furred tongue, anorexia, and constipation. In nervous, irritable individuals and in children, there may be slight delirium, and in very young children during the period of dentition, convulsions may often usher in an attack.

After a day or two of these initiatory symptoms, those characteristic of bronchial catarrh develop.

Pain is experienced beneath the sternum, especially toward its upper part, of a raw, burning, or tearing character, aggravated by a deep inspiration or by coughing; the pain also radiates toward the sides, following the course of the primary bronchial tubes. Tenderness over the sternum is often experienced. Muscular pain and tenderness of rheumatic character are often associated with attacks of bronchitis. Cough is present from the onset, at first in paroxysms of a hard, dry character, changing as the disease progresses, and becoming looser, followed by free expectoration. The expectoration, at first, is small in quantity, almost transparent, frothy, often streaked with blood, and having a salty taste. As the disease progresses it becomes more abundant, of a yellowish or a greenish-yellow color, and of a tenacious consistency. There are present slight fever, hot, dry skin, frequent pulse, loss of appetite, moderate thirst, and constipation. A feeling of languor and weariness, and often considerable depression, quite out of proportion to the febrile state, are not infrequent.

Physical Examination.—On inspection, palpation, and percussion there are no evidences of any abnormal condition. Auscultation, however, reveals, in the early stage, the presence of dry râles, sonorous and sibilant, on both sides of the chest, and harsh breath sounds; in the later stage when expectoration is profuse, moist bubbling râles are heard.

Diagnosis.—The points of resemblance and difference between acute bronchitis and other diseases of the chest will be pointed out when those affections are described. The association of bronchitis with other diseases must not be forgotten.

Prognosis.—Acute bronchitis of the larger tubes usually terminates in complete resolution within two weeks. In children and in the aged, the course is more protracted, and the symptoms more severe, but recovery is the rule. Very aged and feeble persons may rarely succumb.

Treatment.—The patient should be confined to a warm but well-ventilated room, and if aged or extremely young and feeble, placed in bed. Soft diet should be prescribed. A free movement of the bowels should be obtained by the administration of fractional doses of calomel followed by magnesia or some other saline. The action of the skin should be rendered free by the employment of the hot foot-bath, hot drinks, and Dover's powder. During the stage of invasion, quinin sulphate, gr. x (0.6 Gm.), combined with morphin sulphate, gr. 1-6 (0.011 Gm.), will usually prevent or abort an attack. In the *first stage,* in adults, when the mucous membrane is swollen and dry, the sedative expectorants or either of the following prescriptions will give prompt relief:

℞.	Antimonii et potassii tart.....gr. ij	.13	Gm.
	Liquor. ammonii acetatis.....f℥ iv	120.	Cc.
	Spts. ætheris nitrosi ....f℥ j	30.	Cc.
	(Tinct. aconiti, if indicated).fʒ ss	2.	Cc.
	Syr. simplicis ....ad f℥ vj	ad 180.	Cc.

M. S.—Two teaspoonfuls every two or three hours.

Or—

℞.	Vini ipecacuanhæ ......fʒ j	4.	Cc.
	Liq. potassii citrat. .....f℥ iij	90.	Cc.
	Liq. ammonii acetat. ...f℥ iij	90.	Cc.

M. S.—Tablespoonful every two or three hours.

If the cough of the dry stage is severe or if diarrhea follow the use of either of the above combinations, camphorated tincture of opium (paregoric) or codein may be added with advantage, but caution should always be exercised in the use of opium in the dry stage. Tincture of hyoscyamus, ♏x-xv (0.3-1 Cc.), may be employed instead.

For young children, the above combinations in proportionately reduced doses or the following may be used with benefit:

℞. Pulv. ipecac. et opii	gr. v	3. Gm.
Pulv. scillæ	gr. x	.6 Gm.
Hydrargyri chlor. mitis	gr. ij	.13 Gm.
Sacch. lact.	gr. x	.6 Gm.

M.—Ft. chart. No. x.
S.—One every two hours.

The following is an excellent mixture for children:

℞. Potassii citrat.	ʒ ij	8. Gm.
Syr. ipecac.	fʒ ij	8. Cc.
Syr. scillæ	fʒ j	4. Cc.
Syr. limonis	fʒ ij	8. Cc.
Tinct. opii camphorat.	fʒ ij	8. Cc.
Elix. simplicis	ad f℥ iij	ad 90. Cc.

M. S.—Teaspoonful every couple hours.

Locally, in this stage, counterirritation is often of great value. Mustard plasters, or turpentine stupes, or even a few dry or wet cups over the sternum will in many cases serve to relieve the substernal pain and bronchial congestion.

Second Stage: The secretion of the bronchial mucous membrane being copious, stimulating expectorants are indicated, such as ammonium chlorid, ammonium carbonate, squill, potassium carbonate, etc. A reliable combination is:

℞. Ammonii chloridi	ʒ ij	8. Gm.
Scillæ aceti	fʒ iij	12. Cc.
Syr. ipecac.	fʒ ij	8. Cc.
Misturæ glycyrrhizæ comp.	ad f℥ iij	ad 90 Cc.

M. S.—Dessertspoonful every three hours.

Attacks showing a tendency to linger are greatly benefited by the following:

℞.	Terebeni	fʒ ij	8. Cc.
	Creosoti	♏ xxiv	1.5 Cc.
	Mucil, acaciæ	q. s.	q. s.
	Aquæ chloroformi	ad f℥ iij	ad 90. Cc.

M. S.—One teaspoonful every four hours, diluted.

In debilitated individuals, alcohol and strychnin are necessary to overcome the depression. During convalescence these and other tonics, such as iron, quinin, and cod-liver oil are indicated. A change of climate is beneficial.

CAPILLARY BRONCHITIS.

Synonyms.—Broncho-pneumonia (?); "suffocative catarrh."

Definition.—An acute catarrhal inflammation of the mucous membrane of the *terminal* bronchial tubes, or bronchioles; characterized by fever, impeded and increased respiration, impeded circulation, slight cough and scanty expectoration, and symptoms of nonaeration of the blood.

Causes.—It is most common in childhood, following exposure to cold or sudden changes of temperature; may complicate measles, whooping cough, or any of the debilitating diseases. The affection also occurs in the aged. There may be a special microörganism.

Pathologic Anatomy.—Hyperemia, redness, and swelling of the lining membrane of the bronchioles, with the exudation of a tough, tenacious secretion are the principal structural changes.

In those cases in which the air-cells are not involved the air passes, during the act of inspiration, through the secretion blocking the smaller tubes, but is prevented from escaping during the act of expiration, the secretion in the smaller tubes acting as a valve; the result is distention of numerous vesicles, producing a circumscribed or diffused functional emphysema. If the secretion produces complete closure of any of the smaller tubes, the air previously drawn into the vesicles will be absorbed, causing pulmonary collapse (atelectasis).

If the inflammation extends to the alveoli of the lungs, it produces the condition known as *broncho-pneumonia,* a frequent complication in children and feeble elderly people; it is most commonly lobular in character, whence the term "*lobular pneumonia.*"

Symptoms.—Mild bronchitis usually precedes this affection, the onset of which is marked by a rise of temperature, 102°-103°F., increased pulse, increased and difficult respirations, and paroxysmal dyspnea attended with cyanosis. The tongue is coated; the appetite is impaired; the bowels are costive; and headache and restlessness are present. The circulation through the lungs is impeded by the dyspnea, the pulse becomes feeble and flickering, and there results general congestion of the venous system, the countenance becomes livid, the lips and nails blue, the surface cold, and often covered with a clammy perspiration, the mind dull, and in children stupor and convulsions rapidly supervene, the result of the non-aeration of the blood. The cough is slight, but of a suppressed character, and the expectoration scanty, the patient usually swallowing the sputum. When cyanosis occurs, the cough may almost entirely cease; expectoration also ceases, death soon following from apnea and depression.

Physical Signs.—*Percussion* yields a normal note except over those portions of the lungs (a bilateral disease) which are in a state of collapse, in which areas dullness rapidly develops and may as rapidly disappear, changing to other portions of the lungs—shifting dullness.

Auscultation reveals, in the first stage, a feeble, but high-pitched, respiratory murmur, which becomes less distinct and harsh as the disease progresses. The râles in the first stage are fine whistling, and, sibilant, changing in the second stage to fine bubbling or subcrepitant râles. The respiratory murmur is absent over the dull area.

Diagnosis.—There is one point characteristic of capillary bronchitis—it is a general or bilateral disease. Capillary bronchitis is often mistaken for true catarrhal pneumonia, the points of distinction between which will be pointed out when discussing the latter affection.

Prognosis.—In children, on account of their inability to expectorate and consequent tendency to rapid collapse of the lungs, and in the aged, the prognosis is most grave. In the strong and vigorous, recovery follows prompt and energetic treatment.

Treatment.—Supportive measures are indicated from the onset. The patient should be confined to bed and well protected. The temperature of the room should be from 75° to 80°F., and the atmosphere should be moistened with the vapor of boiling water, to which compound tincture of benzoin may be added. The diet should be of the most nutritious character; the great aim being to sustain the powers of life until the catarrhal process has passed through the different stages; hence, milk, eggs, chicken, mutton and beef broths, together with stimulants, should be used from the onset in amounts large enough to overcome the signs of depression, which become manifest early in the attack.

In the first stage, dry cups, mild mustard plasters, and turpentine stupes should be applied to the chest, after which it should be covered with an oiled-silk or cotton jacket. In desperate cases, H. C. Wood advises the alternate use of the hot and cold douche together with the internal administration of stimulating remedies.

High fever may be controlled by hydrotherapy or the use of spirits of nitrous ether, or acetanilid, gr. ii-iv (0.13-0.26 Gm.), in brandy or whiskey. If suffocation is imminent, yellow mercuric subsulphate (turpeth mineral), gr. ii-iv (0.1296-0.2592 Gm.), syrup of ipecac, ʒi (3.70 Gm.), or apomorphin, gr. 1-20 (0.003 Gm.), hypodermically, should be used to induce vomiting, being careful not to repeat the emesis often enough to bring about exhaustion.

For the catarrhal process, potassium or ammonium iodid, gr. i-ii (0.065-0.13 Gm.) every hour for a child, and gr. v-x (0.3-0.6 Gm.), every hour for an adult, is of inestimable value, serving to liquefy or thin the tenacious secretion and to modify the inflammatory changes. Of equal benefit is ammonium carbonate in dose of gr. i-ii (0.065-0.13 Gm.) every hour or two for a child, and gr. v-x (0.3-0.6 Gm.) for an adult. A combination of these remedies is especially useful:

℞. Potassii iodidi	gr. ij	.13 Gm.
Ammonii carbonat.	gr. iij	.2 Gm.
Syr. glycyrrh.	fʒ ss	2. Cc.
Syr. tolu.	fʒ ss	2. Cc.

M. S.—Every two or three hours.

Excellent results have been obtained in the children's wards of the Philadelphia Hospital from the systematic inhalation of oxygen.

Convalescence requires the employment of remedies such as the syrup of hydriodic acid, syrup of the iodid of iron, hypophosphites, cod liver oil, iron, quinin, strychnin, etc.

FIBRINOUS BRONCHITIS.

Synonyms.—Membranous bronchitis; plastic bronchitis; diphtheritic bronchitis; croupous bronchitis.

Definition.—An acute inflammation of the mucous membrane of the larger and middle-sized bronchial tubes, attended with an exudation, forming a membraniform layer, which is closely adherent to the mucous surface; characterized by febrile reaction, cough, difficult breathing, and scanty expectoration, followed by the expulsion of the false membrane in the form of patches or casts.

Causes.—The direct cause is unknown. The affection may be associated with membranous laryngitis, asthma, emphysema, typhoid fever, phthisis, pneumonia, certain skin diseases, or disturbances of menstruation. It may occur in those of feeble health or in tuberculous constitutions, so-called, or it may result from exposure to cold and damp. Spring season, adult life, and male sex are predisposing factors.

Pathologic Anatomy.—The affection begins with hyperemia of the mucous membrane of the bronchial tubes, associated with swelling and edema. Later the surface is covered with a whitish or grayish-white firmly adherent, membranous deposit, cemented together by a coagulable exudation and prolonged from its under surface into the bronchial follicles. Sooner or later it is loosened and detached by a suppurative process and is expectorated after a violent paroxysm of coughing or vomiting. When expectorated, the false membrane, as it has been termed, has either the form of patches, or is thrown off entire from the bronchial tube, and may be found to consist of casts representing, more or less of, the bronchial subdivisions, and presenting an appearance not unlike "boiled macaroni."

On *microscopic examination,* the detached membrane presents fibrillæ which characterize fibrin or lymph in other situations; and if placed in a solution of acetic acid, it becomes greatly swollen, while ordinary mucus contracts and becomes more dense if added to the same solution. Charcot-Leyden crystals, Curschmann's spirals, leukocytes, fat-droplets, and epithelium may be found in the casts.

Symptoms.—There are no symptoms or signs by means of which this variety of bronchitis can be distinguished from ordinary catarrhal bronchitis, prior to the expectoration of the false membrane.

Expectoration is preceded and accompanied by violent paroxysms of coughing, and after more or less of the membrane has been raised, a muco-purulent expectoration, streaked with blood, may be present for several days.

Duration.—The inflammation may be either *acute*, *subacute*, or *chronic*, expectoration of patches or strips of the membrane being repeated at intervals of days, weeks, months, or even years.

Prognosis.—In adults, the outlook is favorable, if not associated with other grave affections, such as phthisis, pneumonia, emphysema. In young children it may cause obstruction to the respiration, and not infrequently proves fatal. The acute form is most serious.

Treatment.—As the character of the inflammation can seldom be determined until the membrane or portions of it have been expectorated, the treatment is at first the same as in attacks of ordinary acute bronchitis.

As soon, however, as the character of the inflammation can be determined, active emesis is the most effective means of removing the obstruction caused by the false membrane, the best agents of this class being yellow mercuric subsulphate (turpeth mineral), apomorphin, ipecac, and zinc sulphate. Inhalations of the vapor of alkaline solutions such as lime-water and solution of sodium bicarbonate, gr. xxx to the fluid ounce (2 Gm. to 30 Cc.), ammonium chlorid, tar (pix liquida), and eucalyptol may also be employed in inhalations. To prevent the formation of the membrane, Bartholow urges the use of ammonium iodid and ammonium carbonate combined, in small doses every two hours. Potassium iodid is also of value. Counterirritation to the chest is of benefit in cases which tend to become chronic. Arsenic and pix liquida should also be given in these cases.

CHRONIC BRONCHITIS.

Synonyms.—Chronic bronchial catarrh; winter cough; secondary bronchitis.

Definition.—A chronic inflammation of the mucous membrane of the larger and middle-sized bronchial tubes; characterized by cough and more or less profuse expectoration, plus, in many cases, the symptoms of emphysema of the lungs. Chronic bronchitis may be either *primary or secondary.*

Causes.—It may follow a succession of acute attacks, or it may be due to exposure to cold and wet or the repeated inhalation of dust, vapors, or other irritants. It is common in the aged. The affection may accompany the infectious fevers, as typhoid fever, influenza, measles, etc., and pulmonic, cardiac, or renal disease, or it may arise indirectly from gout, rheumatism, syphilis, and alcoholism.

Varieties.—I. *Mucous catarrh*, associated with moderate expectoration. II. *Bronchorrhea,* profuse expectoration. III. *Dry catarrh,* scanty expectoration. IV. *Fetid bronchitis.* V. *Bronchiectasis,* or dilatation of the bronchi.

Pathologic Anatomy.—The mucous membrane of the bronchial tube is discolored, being of a more or less dull red, often of a deeply venous hue, mingled with a grayish or brownish color. These changes may be either in patches or extensively diffused. The vessels of the mucous membrane are dilated. The mucous membrane is thickened, resulting in reduction in the caliber of the tube and a roughening of its internal surface. The submucous tissue becomes infiltrated, contracted, and indurated. The elastic and muscular coats of the tubes become hypertrophied, lose their elasticity, and the cartilages become the seat of calcareous deposits.

As the result of the loss of elasticity and muscular tone of the tubes they become irregularly dilated—"*bronchial dilatation.*" The dilatations may be uniform in character, resembling somewhat the fingers of a glove, or they may be sacculated or globular, forming actual cavities in the bronchial structure.

In the *mucous variety* the secretion consists of young cells and mucous corpuscles, having a yellowish color; in the *dry variety,* the "catarrhe sec" of Laennec, or "dry bronchial irritation," the secretion is scanty, tough, semi-transparent, and occurs in globular masses; in *bronchorrhea,* which is usually associated with bronchial dilatation, the secretion is abundant, greenish-yellow in color, and frequently fetid.

The majority of cases of chronic bronchitis are associated with chronic gastric catarrh.

Symptoms.—The most characteristic symptoms of chronic bronchitis are the cough and expectoration. The cough may occur at all hours, but is more severe at night and early in the morning. The cough is not always present. It disappears almost altogether for a time, and then reappears, continuing thus for years. Coated tongue, disagreeable taste, loss of appetite, impaired digestion, with eructations of gases, are present in many cases, due to the chronic gastric catarrh. Unless associated with other diseases, the general health suffers but little, if at all, constitutional symptoms being present only during acute exacerbations.

Mucous catarrh, or, from its occurring most commonly during the winter months, "winter cough," is characterized by paroxysms of cough, more or less violent, followed by the expectoration of a yellowish mucus.

Dry catarrh is characterized by a harsh cough, a feeling of soreness or rawness under the sternum, and the expectoration of small globular masses; this variety occurs with emphysema, gout, rheumatism, and asthma.

Bronchorrhea, which is associated with bronchial dilatation, and most common in the elderly, is characterized by paroxysms of severe coughing, followed by the copious expectoration of greenish-yellow, often fetid, mucus; the amount expectorated often amounts to four or five pints in the twenty-four hours.

Fetid bronchitis, often associated with bronchial dilatation, has an excessively fetid odor of the breath and expectoration. The decomposition of the secretion may cause gangrene of the bronchial mucous membrane, and even of the lung-structure.

Physical Signs.—*Percussion* yields a normal note in simple, uncomplicated cases. In the presence of bronchial dilatation there are diffused spots of the tympanitic or amphoric percussion sound, the physical condition being a circumscribed cavity containing air and communicating with a bronhial tube.

Auscultation reveals the presence of harsh or vesiculo-bronchial respiration associated with more or less profuse, sonorous, sibilant, and large and small bubbling râles; in bronchial dilatation, in addition to the harsh respiration, is found broncho-cavernous breathing, with large and small gurgling râles. Should emphysema complicate chronic bronchitis, the physical signs are somewhat modified, and will be pointed out when discussing that affection.

Diagnosis.—Always examine the urine in case of cough, and particularly in chronic bronchitis, as this condition is one of the most frequent complications of Bright's disease.

Incipient phthisis is often confounded with chronic bronchitis. The diagnosis is not always easy. The physical signs of chronic bronchitis are more or less diffused through both lungs, and not, as a rule, associated with failure of the general health; while in phthisis, from the onset, there is failing health, with a concentration of the physical signs to the apices. The discovery of the bacillus determines the diagnosis.

Bronchiectasis may be distinguished from chronic bronchitis by the paroxysmal coughing, copious expectoration, and physical signs indicating one or more cavities near the base of the lung.

Emphysema is characterized by uniform distention of the chest, dyspnea, hyper-resonance, and feeble expiration.

Asthma is attended by marked dyspnea, hyper-resonance on percussion, dry and moist râles on auscultation, and expectoration of Curschmann's spirals and Charcot-Leyden crystals.

Prognosis.—If unassociated with disease of the lungs, heart, or kidneys, chronic bronchitis is never dangerous to life, although the symptoms are present, more or less, continually, and aggravated upon the least exposure. Rarely is a complete cure recorded.

If associated with phthisis, emphysema, diseases of the heart or of the kidneys, the prognosis is governed by these affections. In turn, it is to be remembered that chronic bronchial catarrh may lead to emphysema of the lungs, bronchiectasis, asthma, or to cardiac dilatation.

Treatment.—In all cases, a careful examination should be made of all the organs to determine whether the affection is primary or secondary. When dependent upon some other disease the greater portion of the treatment should be directed towards the underlying condition. Warmth is beneficial in all cases. The patient should be protected from cold by wearing woolen or silk underclothing the year round, being careful, however, not to clothe to excess. Draughts, wet feet, etc., should be guarded against. A warm atmosphere is especially beneficial, and when possible the patient should be removed to a warm climate. If the expectoration is profuse, a warm dry climate is indicated, but if the expectoration is very scant, the opposite, a moist, warm climate is recommended.

The medicinal treatment has for its object the restoration of the normal tone of the body and the lessening of the local inflammation. The first indication is met by the administration of iron, quinin, strychnin, arsenic, cod-liver oil, etc. In the presence of organic disease special medicinal treatment is required in addition. In cases dependent upon the uric acid diathesis the iodids and alkalies should be administered over an extended period, and the patient should be advised to seek a residence at one of the alkaline springs. When the condition is associated with alcoholism or chronic gastric catarrh, the following combination is of value:

℞.	Ammonii chloridi ......ʒ iij	12. Gm.
	Tinct. nucis vomicæ ....fʒ ij	8. Cc.
	Infus. gentianæ comp., q. s. ad f℥ iv	ad 120. Cc.

M. S.—Dessertspoonful in water before meals.

The bronchial inflammation, itself, calls for the use of stimulating expectorants, prominent among which may be mentioned ammonium chlorid, ammonium carbonate, benzoic acid, balsams of Peru and Tolu, tar, squill, turpentine, oil of eucalyptus, terebene, santal wood oil, cubebs, copaipa, creasote, and terpene hydrate.

For mucous catarrh with acute exacerbations:

℞.	Ammonii chloridi ....ʒ ij	8. Gm.
	Glycerini ....f℥ iss	45. Cc.
	Codeinæ sulph. ....gr. j	.065 Gm.
	Vini picis liq. ....f℥ iij	90. Cc.
	Syr. prun. virg. ....f℥ iss	45. Cc.

M. S.—Tablespoonful every three or four hours.

Dry catarrh is greatly benefited by—

℞.	Potassii iodidi ....gr. v-x	.3-.6 Gm.
	Elix. cinchonæ ....♏xx	1.3 Cc.
	Vini picis liq. ......ad f℥ ss	ad 15. Cc.

M. S.—Three times a day.

For an acute exacerbation of dry or tenacious chronic bronchitis:

℞.	Ammonii chloridi ......ʒ iv	15. Gm.
	Tinct. hyoscyam. ......fʒ iv	15. Cc.
	Syr. scillæ comp. ....fʒ iv	15. Cc.
	Aq. chloroformi ....f℥ ij	60 Cc.

M. S.—One teaspoonful every three hours, diluted.

An excellent expectorant combination in all forms and at any stage of bronchial catarrh is:

℞. Ammonii carbonat. .....gr. xvj	1. Gm.
Ext. scillæ fld. ....fʒ ss	2. Cc.
Ext. senegæ fld. ....fʒ ss	2. Cc.
Tinct. opii camphorat. ..fʒ iij	12. Cc.
Syr. tolu ....f℥ iss	45. Cc.

M. S.—Teaspoonful every few hours, diluted.

Or—

℞. Ext. eucalypti fld. ......℥ i	30. Gm.
Ammonii chlorid ....ʒ i	4. Gm.
Ext. glycyrrhizæ ....ʒ ii	8. Gm.
Syrup Tolutani ....f℥ iii	90. Cc.

M. S.—One teaspoonful every three hours. (Potter).

Or—

℞. Ammonii chlorid. ....gr. xxx	2. Gm.
Tr. opii camph.	
Syr. ipecac ....ad fʒ iii	11. Cc.
Syr. pruni Virg. ....f℥ i	30. Cc.
Syr. Tolutani ...q. s. ad f℥ iv	120. Cc.

M. S.—Teaspoonful every three hours. (Bellevue Dispensary—Dr. Echeverria).

Or—

℞. Acid hydrocyanici dil. .....♏xx	1.23 Cc.
Ammonii carb. ....gr. xl	2.6 Gm.
Syr. Tolutani ....f℥ iv	120. Cc.
Aquæ ....q. s. ad f℥ viii	236. Cc.

M. S.—Four teaspoonfuls every three hours (New York City Hospital—Dr. Welch).

Or—

℞. Syr. scillæ ....f℥ ss	15. Cc.
Tr. opii camph. ....fʒ ii	8. Cc.
Ammoniac. ....ʒ ss	2. Gm.
Syrup. Tolutani ....fʒ x	38. Gm.

M. S.—Teaspoonful as the occasion requires (Potter).

In the bronchorrheal type of the disease, copaipa, ♏v-x (0.3-0.6 Cc.), every three hours, spirits of turpentine, ♏v (0.3 Cc.) every four hours, carbolic acid, gr. ss (0.032 Gm.) four times daily, or the following combination should be administered:

℞. Terebeni ....fʒ ij	8. Cc.
Creosoti ....♏xxx	2. Cc.
Acaciæ ....q. s.	q. s.
Aq. chloroformi ....f℥ j	30. Cc.
Syr. prun, virg. .....ad f℥ iij	ad 90. Cc.

M. S.—Teaspoonful every three or four hours, diluted.

Or—

℞. Copaibæ,	
Syr. Tolutani ....āā ʒ iv	15. Cc.
Spt. etheris nitrosi .....f℥ i	30. Cc.
Aquæ menth. pip. ......f℥ ii	60. Cc.

M. S.—Teaspoonful every four hours (Potter).

In fetid bronchitis, Da Costa recommends the internal administration of carbolic acid, ♏i (0.06 Cc.) every third hour with inhalations of the vapor of water containing carbolic acid, gr. v (0.32 Gm.) to the fluid ounce (30 Cc.), two or three times daily.

The following inhalation may also be used:

℞. Creasote (beechwood)..fʒ i	4. Cc.
Eucalyptol ....fʒ i	4. Cc.
Tr. benzoin comp. .....f℥ ii	60. Cc.

M. S.—Add one teaspoonful to a pint of boiling water and use as an inhalation twice daily.

Locally, counterirritation in the form of flying blisters, or tincture of iodin repeated once or twice a week is of advantage.

ASTHMA.

Synonyms.—Bronchial asthma; spasmodic asthma.

Definition.—A paroxysmal, spasmodic contraction of the muscular layer surrounding the smaller bronchial tubes, and perhaps associated with a tonic spasm of the diaphragm and more or less bronchial catarrh; characterized by spasmodic attacks of distressing expiratory dyspnea, continuing several hours, days, or weeks.

Causes.—The affection is believed to be a true neurosis of the respiratory apparatus. It may result from peripheral or local disturbances in the nervous system. In many cases there is a family history of asthma, chorea, or epilepsy. It is more common in men than in women, and may occur at any age. Atmospheric and climatic changes may act as causes. Some cases are of reflex origin.

Frequently the affection is due to disease of the nasal or bronchial mucous membrane, bronchitis, emphysema, chronic cardiac disease, chronic gastric catarrh, and malarial toxemia. The inhalation of irritating substances such as ipecac, turpentine, dust, etc., may precipitate an attack.

Pathology.—Except in the presence of bronchitis or other affection there are no structural changes. The attacks consist in spasm of the muscular coat with vasomotor turgescence of the mucous coat of the bronchi.

Symptoms.—The onset of a first attack of asthma is abrupt, the succeeding attacks being preceded by prodromes, which the individual rapidly learns to appreciate—to wit.: coryza, bronchial irritation, thoracic constriction, marked dyspepsia, or the scanty passage of pale, limpid urine, the "hysterical urine."

The paroxysm begins, in the majority of instances, in the early morning hours or during the afternoon, with a feeling of anguish and constriction in the chest and an intense desire for air. The breathing is accompanied with loud wheezing, the face is flushed, at times even cyanosed and bathed in perspiration, the eyes staring, the eyeballs protrude, and the muscles of the neck become prominent as they aid in the effort for air. The dyspnea soon becomes so severe that the inspiration is but a gasp, the lips are pallid, cyanosis deepens, and the patient feels as if death were impending. Owing to the tonic contraction of the smaller bronchi, the air drawn into the alveoli escapes imperfectly, resulting in the expiratory dyspnea, the emphysematous chest, and the lowered position of the diaphragm. During the paroxysm there is a short, dry cough, becoming more loose as the attack subsides.

After some minutes or hours the respiration becomes easier, the air in the lungs changes, the cyanosis disappears, and gradually the paroxysm ceases, the patient feeling exhausted and the chest fatigued.

The *sputum* of asthma is unique. Early in the paroxysm it is raised with difficulty, and takes the form of rounded gelatinous masses ("perles" of Laennec). If these pellets are carefully examined, they will be found to consist of molds of the smaller bronchi, and, under the microscope, show Leyden's crystals and Curschmann's spirals. After a day or two the sputum becomes muco-purulent, and the spirals and crystals are absent.

The duration of an attack varies from one to many hours, or even days. Instead of single paroxysms, slight remissions may occur at intervals of one, two, or three hours, to be followed by exacerbations lasting from four to six hours, continuing for a week or two, preventing the patient lying down or taking food.

Physical Signs.—*Inspection* shows marked dyspnea, with distention of the chest.

Percussion yields, during the paroxysm, hyper-resonance or a vesiculo-tympanic note (the band-box tone of Bamberger) over both lungs, due to the retained air in the alveoli.

Auscultation during the first stage reveals a feeble or absent vesicular murmur, with prolonged expiration, associated with loud, wheezing, whistling, sibilant, and sonorous râles; as the paroxysm subsides, the vesicular breathing becomes more noticeable, and is accompanied by moist râles.

Prognosis.—The disease is essentially chronic and recovery seldom occurs, except when due to reflex causes that may be removed. The paroxysms may be relieved by treatment. In itself asthma is not fatal to life; but if the paroxysms are frequently repeated, there results either emphysema, cardiac dilatation with subsequent dropsy, or even cerebral hemorrhage.

Attacks of asthma frequently occur as a complication in emphysema, chronic bronchitis, valvular diseases of the heart, and Bright's disease.

Treatment.—There are two indications to meet: the relief of the paroxysm, and prevention of its recurrence.

To relieve the paroxysm, no medication is so effective as the hypodermic injection of morphin sulphate, gr. ⅙-¼ (0.011-0.016 Gm.), combined with atropin sulphate, gr. 1-100 (0.00065 Gm.). Chloral, gr. x (0.6 Gm.) in the absence of cardiac complications is very beneficial. Inhalation of chloroform or a few drops of amyl nitrite will also serve to relieve the paroxysm. Drinking of strong hot black coffee or the administration of caffein citrate, gr. iii-v (0.2-0.3 Gm.), hypodermically, in a cachet, or in solution, is of great value.

The following combination by hypodermic injection is often most successful in relieving an attack of asthma, and particularly if complicated with cardiac or nephritic disease, continuing the combination after relief, in pill form or solution, at ordinary intervals for several days:

℞.	Spirit. glonoini ....♏ ij	.12 Cc.
	Strychninæ sulph. ....gr. 1-50	.0013 Gm.
	Morphinæ sulph. ....gr. 1-20	.003 Gm.

M. S.—One dose. For hypodermic use.

Page strongly recommends sodium nitrate, as in the following formula:

℞.	Pulv. sodii nitritis ......gr. xxiv	1.6 Gm.
	Aquæ ....f℥ j	30. Cc.

M. S.—Teaspoonful at once; repeat in half an hour, once or twice if necessary.

Dr. Pepper speaks highly of the following for the paroxysm:

℞.	Ammonii bromidi ....ʒ ij ℈ij	10. Gm.
	Ammonii chloridi ....ʒ iss	6. Gm.
	Tinct. lobeliæ ....fʒ iij	12. Cc.
	Spts. ætheris comp. ....f℥ j	30. Cc.
	Syr. acaciæ ....q. s. f℥ iv	ad 120. Cc.

M. S.—Dessertspoonful in water every hour or two, diluted.

The nauseating expectorants, such as lobelia, ipecac, and squill, are at times of value. Fluid extract of grindelia, ♏xx (1.3 Cc.) repeated every two hours, is sometimes useful. Inhalations of the fumes of belladonna, stramonium, nitre paper, or ethyl bromid, or the use of the various pastilles or cigarettes, are of great benefit in many cases. A twenty percent solution of menthol, and oxygen have also been employed in the same manner with success.

After the employment of many drugs I have finally come to depend upon potassium iodid, gr. v-x (0.3-0.6 Gm.), every three hours, either alone or combined with tincture of belladonna, ♏v (0.3 Cc.), or nitroglycerin, gr. 1-200 to 1-100 (0.00032-0.00065 Gm.). Another valuable remedy is the syrup of hydriodic acid, ʒss-ʒi (2-4 Cc.) every three hours diluted. If an attack is impending, it may often be aborted by drinking freely of strong, black coffee, or by full doses of the bromids.

Bartholow employs the following in cigarettes:

℞.	Sodii arseniat ....ʒ ss	2. Gm.
	Aquæ destillat ....℥ i	30. Cc.

M. S.—Moisten unsized white paper, and roll into cigarettes, each containing gr. ¼ t gr. i of the salt. Two or three of these should be inhaled daily.

Trousseau's cigarettes are:

℞.	Belladonnæ fol. ....ʒ i	4.	Gm.
	Stramonii fol.		
	Hyoscyami ....āā ʒ ss	2.	Gm.
	Ext. opii ....gr. iii	.194	Gm.
	Aquæ laurocerasi ....q. s.		

M.—Dissolve the opium in the water and moisten the leaves therewith. When dry roll into twelve cigarettes. Smoke two a day.

Potter recommends the following prescription:

℞.	Ext. stramonii ....gr. ii	.130	Gm.
	Potass. iodid. ....ʒ iss	6.	Gm.
	Ammonii carbonat. .....ʒ i	4.	Gm.
	Tr. lobeliæ ....fʒ iss	6.	Cc.
	Aquæ chloroformi.q.s. ad f℥ viii	480.	Cc.

M. S.—Tablespoonful every six hours.

The following combination may be of service:

℞.	Morphin. sulphat. ....gr. ss	.032	Gm.
	Ext. belladonnæ fld. ....fʒ ii	8.	Cc.
	Ext. grindeliæ robust fld..fʒ ii	8.	Cc.
	Spt. etheris comp. ......fʒ iv	15.	Cc.
	Syrup. ....q. s. ad f℥ ii	60.	Cc.

M. S.—Teaspoonful as the occasion requires (Kings County Hospital).

During the interval between the attacks, the nasal mucous membrane should be carefully examined and in the presence of morbid conditions should receive appropriate treatment. The condition of the heart and lungs should likewise be ascertained. The digestive tract should also receive attention. The various reflex conditions that may induce the paroxysms should be removed. Dry climate is usually most beneficial. The long-continued administration of potassium iodid and arsenic is of special value. As additional aids may be mentioned systematic exercise short of fatigue, bathing, regulated diet, and, when possible, a change of climate.

HAY ASTHMA.

Synonyms.—Hay fever; autumnal catarrh; rose cold.

Definition.—An acute, specific, catarrhal inflammation of the upper air-passages, extending to the bronchial tubes, associated with spasmodic contraction of their muscular layer, occurring at a particular season of the year, characterized by coryza, croupy or wheezy cough, and difficult respiration.

Causes.—The nervous system seems especially predisposed in many cases. Heredity, sedentary life, uric acid diathesis, nasal disease, and neurotic constitution are important etiologic factors. The disease becomes manifest in the spring and autumn, and the attacks may be brought about by the inhalation of irritating dusts or vapors, or the pollen of grasses, rye, corn, wheat, or roses. The affection is encountered most frequently in the cities and in low countries.

Pathology.—A hyper-sensitiveness of the nasal mucous membrane is believed to be the only change. Associated with this, however, it is rather common to find hypertrophic rhinitis, enlargement of the inferior and middle turbinated bones, nasal polyps, and deflection of the nasal septum, the relief of which conditions is often followed by cure of the hay fever.

Symptoms.—The attack begins with irritation of the eyes, coryza, and sneezing, with a clear watery nasal discharge. The congestion extends to the eustachian tube and to the larynx and bronchial tubes, thereby inducing a hoarse, croupy, and wheezing cough, with difficulty in breathing. The dyspnea occurs in paroxysms, which are often as severe as those occurring in true asthmatic attacks. Mild nervous depression is usually present. The paroxysms remit after a few days, to recur after an interval of several days or weeks, and to be followed by another remission, and so on until the season changes. The bronchial catarrh persists during the entire attack. Constitutional symptoms are mild in the absence of complications.

Complications.—Capillary bronchitis, congestion or edema of the lungs, or pneumonia may occur as complications.

Duration.—Unless a change of climate is resorted to, paroxysms of hay fever continue more or less severe for six, eight, or ten weeks of the year, each year the paroxysms growing more severe.

Prognosis.—The affection never proves fatal in itself, but one or more of the following sequelæ may result, to wit.: asthma, chronic bronchitis, or loss of the special sense of hearing or of smelling.

Treatment.—There is no specific. In those cases in which nasal disease exists, considerable relief may be afforded by attention to the nasal channels.

An attack of hay asthma is often prevented by a change of climate during the season of the year when the attacks are most common, to wit.: the early autumn. Any of the following locations may be selected—White Mountains, Catskills, Adirondacks, Rocky Mountains, or a sea voyage. Certain seaside resorts, particularly Long Branch, Beach Haven, Fire Island, Nantucket, and Mount Desert, seem to be especially beneficial to hay-fever patients.

The condition of the general health should receive very close attention. Many patients are more or less run down and require tonics, such as iron, strychnin, arsenic, quinin, phosphorus, etc., over an extended period.

℞. Liq. potass. arsenit. ....fʒ i	4. Cc.
Syr. hypophos. ..q. s. ad f℥ iv	120. Cc.

M. S.—Two teaspoonfuls after meals.

Or—

℞. Ext. belladonnæ	
Ext. cannabis Indicæ .āā gr. i	āā .065 Gm.
Camphor ....gr. xv	1. Gm.
Quinin sulphatis ....gr. xx	1.296 Gm.

M.—Disp. in capsul. No. vi.
S.—One every three hours.

The digestive tract should be carefully examined. Indigestion should receive prompt treatment. Constipation should be avoided. Fruits and vegetables should form the greater portion of the diet, and animal foods, coffee, and tea, should be interdicted. All the avenues of excretion should be maintained in their normal condition. Frequent hot baths, massage, electricity, diuretics, and diaphoretics may be employed with this end in view.

The attacks may apparently be aborted at times, by internal treatment. Quinin sulphate, gr. v (0.3 Gm.), three times daily, administered one month before the attack is expected, has been successful. Dover's powder, gr. v (0.3 Gm.), three times daily, or the following may be used for the same purpose:

℞.	Atropinæ sulph.	gr. ⅕	.012 Gm.
	Morphinæ sulph.	gr. ¼	.016 Gm.
	Strychninæ sulph.	gr. ⅛	.008 Cm.
	Quininæ muriat.	gr. x	.65 Gm.
	Sodii arseniat.	gr. ⅙	.011 Gm.

M.—Ft. pil. No. xxx.

S.—One every hour until dryness, then two or three hours apart.

The following is of benefit during the attack:

℞.	Ext. hyoscyami	gr. xii	.775 Gm.
	Potass. iodid	ʒ i	4. Gm.
	Potass. bicarb.	ʒ ii	8. Gm.
	Ext. glycyrrhizæ puræ...	ʒ iv	15. Gm.
	Aquæ anisi	f℥ ivss	136. Cc.

M. S.—Dessertspoonful every four hours until relieved (Weber).

Local Treatment.—Dr. W. C. Hollopeter reports wonderful success in over two hundred patients with the following plan of treatment:

"For the last ten years I have used the ordinary Dobell's solution. This I thoroughly use in both nostrils, first by means of a hand-ball atomizer, after which, with a curved aluminum applicator, I very carefully swab the whole naso-pharynx. I scrub most carefully every portion of the mucous membrane, being sure to reach between the turbinated bones and all around and over every slight prominence. I then as carefully dry the membrane with clean cotton, and use freely blandine comp. (a mild solution of menthol in albolene), loosely plugging the nose for a few minutes to retain the oily application. Great stress is laid upon the thoroughness of the application and the correction of any ills the patient may have."

The application of tablets of cocain hydrochlorate, gr. ⅙ (0.011 Gm.), or the same drug in four percent solution every two or three hours will afford great relief. The possibility of contracting the cocain habit from this treatment should not be overlooked. A much safer plan is to apply pledgets of cotton soaked in a solution of adrenalin hydrochlorid (1 to 4000). Bartholow advises the thorough application of quinin to the nares. The following application is also of value:

℞. Menthol	ʒ j	4. Gm.
Cerat. simpl.	℥ ij	60. Gm.
Ol. amygd. dulcis	f℥ iss	45. Cc.
Zinci oxidi puræ	ʒ j	4. Gm.
Acid. carbolici	ʒ ss	2. Gm.

M. S.—Apply every two hours.

Or—

℞. Tinct. iodin.	fʒ i	4. Cc.
Acid. carbol.	gtt. x	.65 Gm.
Aquæ destillat.	f℥ iv	120. Cc.

M. S.—For local use with atomizer or post nasal syringe (Potter).

EMPHYSEMA.

Synonym.—Vesicular emphysema.

Definition.—Dilatation of or increase in the size and capacity of the air-vesicles, characterized by enlargement or distention of the lungs, difficulty of breathing, especially on exertion, and associated sooner or later with dilatation of the heart.

Causes.—The predisposing cause of emphysema is a hereditary nutritive derangement of the lung-structure, often associated with a rigid enlargement of the thorax.

The exciting cause is either too forcible and long-continued inspiration,—*the theory of inspiration*,—or the excessive mechanical distention of the vesicular walls by forced expiration—*the theory of expiration*. But for either of these theories to be operative the lung-structure must be congenitally weak, for if violent respiratory efforts alone were the essential factor, the disease would be much more frequent.

What is known as *vicarious* or *compensatory emphysema* is a distention of the air-cells of the healthy portion of the lung, some other part being the seat of consolidation.

Interlobular emphysema is the presence of air in the spaces between the lobules of the lungs underneath the pulmonary pleura.

Ordinary vesicular emphysema is known also as *pseudo-hypertrophic emphysema* on account of the increase in the capacity of the vesicles, due to distention. The walls of the vesicles are atrophic to a greater or less extent.

Senile emphysema is another variety; often termed "small-lunged emphysema." There is true atrophy of the pulmonary vesicles, although their capacity may be relatively increased.

Pathologic Anatomy.—The situation of vesicular emphysema is, in the majority of cases, the superior portions of the chest, and is more marked on the left side than on the right.

An emphysematous lung feels remarkably soft to the touch, and upon cutting a dull, creaking sound is barely perceptible. It is of a pale-red color; the vesicular walls are thinner and slighter; the vesicles are greatly enlarged, sometimes to the size of a pea or bean, and have an irregular shape, and traversing most of these large sacs (dilated vesicles) a few delicate bands, the remains of the lacerated interalveolar septa, are visible. With the destruction of the septa many of the capillaries are destroyed, leaving the emphysematous tissue remarkably bloodless and dry. In consequence of the destruction of so many of the capillaries, the obstruction to the pulmonary circulation becomes so great that the pulmonary artery and right cavities of the heart are greatly distended; finally the muscular tissue of the heart undergoes granular, followed by fatty, degeneration. The distention of the veins results in a general venous stasis, to wit.: nutmeg liver, congested kidneys, and gastrointestinal catarrh.

Symptoms.—The disease is often not suspected until it is well developed. The chief symptoms of vesicular emphysema are difficulty of breathing (dyspnea), greatly aggravated on exertion; more or less cough, the result of an attending bronchitis, and the various symptoms resulting from dilatation of the heart, particularly cyanosis without marked distress. The discomfort of the patient is often increased by paroxysms of asthma.

Physical Signs.—*Inspection.* The shoulders are rounded, the intercostal spaces widened, and the vertical diameter elongated, with circumscribed prominences between the clavicles and nipples, often increased by the act of coughing—the peculiar "barrel-shaped" chest, characteristic of this disease. The character of the respiratory movements is marked, there being but slight movement observed on forcible respiration, the chest having the constant appearance of a full inspiration.

Palpation.—The vocal fremitus is diminished, and the cardiac impulse depressed and nearer to the sternum.

Percussion.—The resonance is increased (hyper-resonant) over all the emphysematous portions, and, if the whole lung be involved, extends to the seventh or eighth rib anteriorly and to the twelfth rib posteriorly. The hepatic dullness may not begin until the inferior margin of the ribs is reached; the cardiac dullness is lessened, on account of the emphysematous lung nearly covering the heart.

Auscultation.—The vesicular murmur is weakened, and in pronounced cases almost absent. If bronchitis be present, the inspiratory sound may be rough or sibilant in character, but its duration is always shortened. Expiration is always prolonged, and if bronchitis be present, may be associated with more or less pronounced moist or bubbling râles. The first sound of the heart is lessened in intensity and duration, the second sound being sharply accentuated.

Diagnosis.—*Bronchitis* is distinguished from emphysema by the absence of dyspnea, hyper-resonance of the chest, changes in its shape, size, and movements, and the disturbance of the circulation.

Spasmodic asthma, by the paroxysmal character of the affection, emphysema being a permanent malady, with attacks of asthma.

Cardiac diseases due to other causes than emphysema do not have the characteristic physical signs of that affection.

Prognosis.—Vesicular emphysema is essentially a chronic disease. In itself it rarely proves fatal, but if aggravated from any cause, or if associated with frequent or prolonged asthmatic paroxysms, the cardiac changes are hastened, and general dropsy supervenes, death occurring from exhaustion, or, more commonly, as the result of intercurrent attacks of pneumonia.

Treatment.—It being impossible to restore the altered lung-structure, the indications for treatment are to relieve the symptoms and to endeavor to prevent its further progress.

For the relief of the asthmatic paroxysms, morphin sulphate, combined with atropin sulphate, may be used hypodermically, or fluid extract of quebracho, f℥ss-i (2-4 Cc.), may be given every hour until benefit is afforded. Caffein citrate, gr. ii-v (0.13-0.3 Gm.) alone or in combination with nitroglycerin, strychnin, or morphin, potassium iodid, or inhalations of oxygen, may be employed for the same purpose.

For the attacks of bronchial catarrh the following is of value:

℞. Ammonii chloridi	ʒ ij	8. Gm.
Tinct. hyoscyami	fʒ iv	15. Cc.
Glycerini	f℥ j	30. Cc.
Syr. prun. virg.	ad f℥ iv	ad 120. Cc.

M. S.—Half-tablespoonful every few hours, well-diluted.

To *prevent the progress of the affection,* remove the bronchial catarrh, relieve the difficulty of breathing, and strengthen the cardiac action; no one combination seems comparable with the following for this purpose:

℞. Potassii iodidi	gr. v	.3 Gm.
Strychninæ sulph.	gr. 1-32	.002 Gm.
Liq. potassii arsenit.	♏ v	.3 Cc.
Aq. lauro-cerasi	fʒ j	4. Cc.

M. S.—Four times a day, well-diluted.

But of all means hitherto proposed for the relief of emphysema, nothing has approached the inhalation of compressed air, by means of the apparatus of Waldenberg. For attacks of cyanosis a free venesection often saves life, combined with and followed by full doses of spirits of glonoin (nitroglycerin). The dropsy arising from failure of the heart to compensate for the circulatory derangement in the lungs, may be relieved for a time, by the use of digitalis and strychnin sulphate, or caffein citrate, the last two being cardiac and respiratory tonics and stimulants, and the last mentioned also a diuretic.

HEMOPTYSIS.

Synonyms.—Bronchial hemorrhage; broncho-pulmonary hemorrhage; bronchorrhagia.

Definition.—The expectoration of pure or unmixed blood, usually of a bright red color, following the act of coughing.

Causes.—In the majority of cases, it is the result of tubercular deposition in the walls of the minute bronchial arteries, excessive cardiac action, bronchial congestion, or excessive bodily exertion, straining, lifting, or running. It may also be due to traumatism, pulmonary congestion, gangrene, infarction, or cancer, ulceration of any portion of the respiratory tract, or to rupture of an aneurysm. In very rare instances it may be produced in the course of hemophilia, purpura, or scurvy, and may be an example of vicarious menstruation. Cases may occasionally be observed in which no cause can be detected.

Pathologic Anatomy.—Hemoptysis rarely causes death in itself, so that few opportunities for observing post-mortem appearances are obtained, and when they do occur, the location of the hemorrhage is seldom found. The air-passages are more or less filled with clotted blood; the mucous membrane is swollen, and of a dark-red color; rarely, pale and bloodless. The air-cells contain blood clots, or are distended with air, the bronchi being filled with clots, preventing its escape. Unless the clots are rapidly removed by expectoration or absorption, a secondary inflammation develops around about them.

Symptoms.—"Spitting of blood" occurs suddenly; rarely, it is preceded by epistaxis, cardiac palpitation, and some difficulty of breathing. It begins with a sensation of warmth under the sternum, tickling in the throat, a sweetish taste in the mouth, an attempt to remove which by the act of coughing is followed by a warm, saltish, bright red, frothy liquid gushing from the mouth and nose. The blood is alkaline in reaction and mixed with air and mucus. The quantity of blood raised varies from an ounce to a pint. The appearance of the blood depresses the individual, he becoming pale, tremulous, often fainting. The attack may subside within half an hour to several hours, returning for several days, in the meantime the expectoration being either bloody or streaked with blood. A slight febrile reaction, with chest pains, supervenes upon the hemorrhage, the result of the inflammation at the site of the bleeding, which soon subsides, except when blood-clots develop a secondary pneumonia, which may undergo the cheesy metamorphosis.

Auscultation reveals the presence of coarse, bubbling râles in circumscribed areas of the chest.

Diagnosis.—From *epistaxis*, or hemorrhage from the posterior nares, it is distinguished by the absence of air-bubbles and an inspection of the fauces and the nasal cavities.

Hematemesis, or hemorrhage from the stomach, differs from hemoptysis in the blood being vomited instead of expectorated, of a dark color, clotted, mixed with the acid contents of the stomach, followed with black, tar-like stools, and the absence of râles in the chest.

Exceptions to the above occur when the blood from the lungs is first swallowed and afterward raised by vomiting, or when the hemorrhage in the stomach is caused by the erosion of a large artery, the result of ulcer of the stomach; in these cases, however, the raising of blood is preceded by epigastric pain and the blood is not frothy.

Prognosis.—Hemoptysis, in itself, rarely terminates fatally, except in advanced phthisis and aneurysm, although causing much depression; the patient rapidly recovers, unless secondary pneumonia results.

Treatment.—Perfect rest in bed, with the head and shoulders elevated and absolute quiet is essential. The diet should be bland and unirritating, and the drinks cool, the patient being allowed to slowly swallow small particles of ice. An ice-bag placed over the chest, if it does not cause chilliness, is sometimes of value. Common salt, slowly dissolved in the mouth, is a popular remedy, and, while of little or no benefit, serves to occupy the attention of the patient and friends until medical advice is obtained. The hypodermic injection of morphin sulphate, gr. ¼ (.016 Gm.), combined with atropin sulphate, gr. 1-60 (0.001 Gm.), will usually control a hemorrhage immediately. The officinal one percent solution of nitroglycerin in half minim to minim doses every half hour, often promptly checks the hemorrhage. The intrapulmonary pressure may be lowered and the flow of blood consequently lessened by the application of firm ligatures to the limbs. In protracted cases, saline purgation may be of benefit. The following prescription may also be employed:

℞. Acidi gallici	gr. xv	1. Gm.
Acidi sulphurici dil.	♏ x	.6 Cc.
Aquæ cinnamomi	fʒ iv	15. Cc.

M. S.—One dose; to be given every fifteen or twenty minutes.

Other drugs, such as tincture of matico, fʒi (4 Cc.), fluid extract of hamamelis, ♏xx-fʒi (1.3-4 Cc.), alum, gr. xx (1.3 Gm.), gallic acid, gr. v-x (0.3-0.6 Gm.), and oil of turpentine, ♏v-xv (0.3-1 Cc.), frequently repeated, have been used with success.

The hypodermic injection of ergotin, gr. x-xxx (0.6-2 Gm.), or the internal administration of fluid extract of ergot, ʒss-j (2-4 Cc.), are also recommended, but I consider them injurious.

Inhalations, by means of the steam atomizer, of either Monsel's solution, or tincture of the chlorid of iron, may be of value when other means have failed. Da Costa advises, for frequent small hemorrhages recurring daily, a combination of cupric sulphate, gr. 1-12 (0.005 Gm.), and extract of opium, gr. 1-12(0.005 Gm.), repeated as the occasion requires.

Bartholow employs the following:

℞.	Plumbi acetat. ....gr. xx	1.3	Gm.
	Pulv. digitalis ....gr. x	.65	Gm.
	Pulv. opii ....gr. v	.324	Gm.

M.—Ft. pil. No. x.
S.—One every four hours.

The following formula may be used at times:

℞.	Aluminis ....ʒ i	4.	Gm.
	Sacch. alb. ....ʒ ss	2.	Gm.
	Pulv. ipecac. comp. ....gr. xx	1.3	Gm.

M.—Ft. pulv. No. vi.
S.—One powder every two hours (Skoda).

DISEASES OF THE LUNGS.

CONGESTION OF THE LUNGS.

Synonyms.—Pulmonary engorgement; hypostatic congestion.

Definition.—An increase in, or abnormal fullness of, the capillaries of the air-cells; *active* congestion when the result of an accelerated circulation; *passive* congestion when caused by an impeded outflow from the capillaries.

Causes.—*Active.* Increased cardiac action; over-exertion; alcoholic excesses; mental excitement; inhalation of cold or hot air.

Passive.—Obstruction to the return circulation. Dilated heart; valvular diseases; low fevers (hypostatic congestion); Bright's disease.

Pathology.—The congested, or engorged lung, has a bloated, dark-red appearance; its vessels are distended to the uttermost, the tissues succulent and relaxed, blood flowing freely over the cut surface; a bloody, frothy liquid is present in the bronchi, and the alveolar walls are so much swollen that the condensed lung shows scarcely any indication of its cellular structure, resembling the tissue of the spleen (*splenification*).

Symptoms.—*Active congestion* precedes inflammatory pulmonary conditions, and is characterized by rapidly developing thoracic distress and difficult breathing, flushed face, strong, full pulse, throbbing carotids, cardiac palpitation, congested eyes, and a short, dry cough, followed by scanty, frothy expectoration, slightly streaked with blood. The presence of fever indicates subsequent inflammation or pneumonia.

Passive congestion develops slowly, with difficulty in breathing, blueness of the body-surface, and an almost continuous hacking cough, followed by scanty, blood-streaked, expectoration.

Physical Signs.—*Percussion* shows the resonance of the lungs slightly diminished, the quality of the sound being somewhat tympanitic. *Auscultation* reveals diminution of the vesicular murmur and the presence of sub-crepitant râles.

Duration.—Active congestion lasts from three to five days, terminating in resolution, hemorrhage, or pneumonia. The onset may be so severe and overwhelming that death rapidly supervenes. Passive congestion develops slowly and is subject to many and great variations, depending on the cause.

Diagnosis.—Active congestion of the lungs cannot be distinguished from the stage of engorgement of a true pneumonia.

Prognosis.—An acute congestion of the lungs may prove fatal within a few hours, but under prompt treatment it generally terminates favorably.

The passive form is controlled entirely by the cause.

Treatment.—In *active congestion* in strong and vigorous individuals, ice-bags and wet cups applied to the chest, or venesection are of value. The internal administration of tincture of aconite, ♏i-ii (0.06-0.12 Cc.) every half hour, and saline cathartics is also beneficial in such cases. Rest in bed is essential in all cases.

In *passive congestion* in addition to treatment directed towards the underlying cause, there should be dry or wet cups applied to the chest, and hydragogue cathartics, digitalis, and strychnin administered. If much depression is present, stimulants are indicated.

EDEMA OF THE LUNGS.

Synonym.—Pulmonary edema.

Definition.—An exudation of serum into the pulmonary interstitial tissue and the alveoli of the lungs; characterized by dyspnea, cough, and a frothy, blood-streaked expectoration.

Causes.—Pulmonary edema is the result of stasis, occurring when the outflow of venous blood in the lung meets an obstacle that cannot be overcome by the right ventricle, as in cardiac diseases, in which the left ventricle fails, Bright disease, and alcoholic excesses, causing cardiac depression. It may be a sequel to other lung inflammations.

Pathologic Anatomy.—The lung-tissue is swollen, and does not collapse when the chest is open. The elasticity of the tissue has disappeared, and it pits upon pressure. If following acute congestion of the lungs, the color is red; if a symptom of a general dropsy, its color is pale. On cutting into the edematous spots, an enormous quantity of albuminous fluid, sometimes clear, at other times of a red color, mixed more or less with blood, flows over the cut surface. The liquid is filled with bubbles, is frothy, from being copiously mixed with air, providing the air-cells have not been entirely filled with serum, thereby excluding the air.

Symptoms.—The pre-eminent symptom is dyspnea, the breathing being hurried, labored, and rattling, all the accessory muscles of respiration being called into action. The sense of oppression and anxiety is extreme. There is also a constant, harassing, short cough, and the expectoration is a blood-streaked, frothy mucus. The action of the heart may be tremulous or feeble. The face is at first flushed, but as the left ventricle fails, or if the effusion into the air-cells be sufficient to prevent the entrance of air, symptoms of cyanosis rapidly supervene, the pulse becoming feeble, the surface cold, the breathing shallow and hurried, and the cough suppressed, stupor replacing the restlessness, soon deepening into coma.

Physical Signs.—*Percussion* reveals no change in the percussion note in the absence of other lung diseases except slight impairment. *Auscultation* demonstrates weak breath-sounds, and subcrepitant and bubbling râles.

Diagnosis.—*Acute pneumonia* in the earlier stages is the only condition likely to be confounded with edema of the lungs, but as the two diseases progress, the picture of pulmonary edema is so characteristic that it cannot be mistaken.

Prognosis.—Grave, and particularly if occurring in pneumonia, cardiac, or Bright's disease. In the majority of instances it is a terminal symptom coming on in all forms of acute and chronic diseases.

Treatment.—As a rule remedies are useless. The indication is to maintain the heart, and this may be produced by the hypodermic injection of atropin sulphate, gr. 1-60 (0.001 Gm.), repeated as necessary, strychnin sulphate, gr. 1-24 (0.0035 Gm.), repeated every half hour, caffein citrate, gr. iii-v (0.2-0.3 Gm.), spartein sulphate, gr. i-ii (0.065-0.13 Gm.), every hour or two, or digitalin, gr. 1-60 to 1-30 (0.001-0.002 Gm.), every two hours. Two or more of these drugs may be combined with advantage. Occasionally relief follows a free venesection or the application of wet cups to the chest. Purgation with hydragogue cathartics is a useful adjunct to the treatment. Alcoholic stimulants and ammonia are also valuable. Counterirritation, ice poultices, hot foot-baths, diuretics, and inhalations of oxygen may be employed.

CROUPOUS PNEUMONIA.

Synonyms.—Lobar pneumonia; pneumonitis; fibrinous pneumonia; pleuro-pneumonia; lung fever; winter fever.

Definition.—An acute, infectious, croupous inflammation, involving the vesicular structure of the lungs rendering the alveoli impervious to air; characterized by a severe chill, headache, fever, thoracic pain, dyspnea, cough, rusty sputum, and great prostration.

Causes.—Croupous pneumonia is an infective disease caused by the *Diplococcus pneumoniæ* of Fränkel, "which has its seat of election in, and produces its chief effects on, the lung." The microorganism is found in the sputum and in the lungs in the majority of cases. "Occasionally other microörganisms seem to occasion typical fibrinous pneumonia. Among these are the pneumococcus of Friedländer, streptococci, staphylococci, the bacillus of typhoid fever, the bacillus of influenza, and the bacillus coli communis. In some cases in which bacteria other than the diplococcus are supposed to be the cause there is doubtless mixed infection, but it must be accepted at the present time that a number of microörganisms are capable of causing the disease." (Stengel.)

All ages are liable. Males are more frequently affected than females. One attack predisposes to another. Debilitating conditions render individuals more susceptible. Alcoholism is among the most frequent predisposing factors. The affection is most frequent in winter, at times occurring epidemically, the result of atmospheric conditions, and exposure to draughts and cold. Gout, rheumatism, diabetes, and Bright's disease may also be causes.

Pathologic Anatomy.—The most frequent seat of croupous pneumonia is the lower right lobe; the next most frequent seat is the lower left lobe; the next, the upper right lobe, although in children and the aged this lobe is affected equally as often as the right lower lobe.

The changes are: I. *Hyperemia* (engorgement); II. *Exudation* (red hepatization); III. *Resolution* (gray hepatization); or the lung may undergo purulent transformation with the development of abscesses (yellow hepatization).

I. *Stage of hyperemia,* or congestion consists in distention of the vessel of the alveoli encroaching on the cavity of the air-vesicle; the lung has a reddish-brown color, is more resistant, and is heavier, sinking somewhat lower in water than a normal lung, and having a slight exudation upon the vesicular surface. The same changes are seen in the adjacent bronchioles.

II. *Stage of exudation* consists in the exudation of a viscid, fibrinous fluid, mixed with white and red corpuscles and blood, which rapidly coagulate, firmly enclosing the corpuscles and completely filling the alveoli. When the exudation and coagulation are completed, the lung is red, sinks at once when placed in water, and its elasticity is destroyed. When cut, the color, density, and granular appearance so closely resemble the cut surface of a section of the liver that Laennec termed the condition *red hepatization.* A thin section shows under the microscope, as a rule, the lancet-shaped diplococcus of Fraenkel, as well as staphylococci and streptococci.

III. *Resolution,* or gray hepatization follows in the majority of cases, the coagulated albuminous exudation undergoing liquefaction and absorption, the cellular element undergoing a fatty degeneration, the greater part being absorbed, the remainder expelled during acts of expectoration, the alveoli returning to their normal condition, as to capacity, function, and elasticity. The consolidated area softens and becomes mottled gray in appearance.

If resolution be retarded and portions of the coagulated exudation undergo *purulent transformation* changing from a yellowish to a greenish-yellow color (yellow hepatization), pus cells are rapidly formed, the part becoming a granular, fatty mass. The portions of the lung not undergoing this purulent transformation retain the reddish color with intermixed yellowish patches. The purulent contents may be ejected in part, the remainder undergoing fatty degeneration and finally absorption.

Abscess of the lung may result from the lung-structure becoming involved in the purulent disintegration. Abscesses may be solitary or in great numbers, which by disintegration of intervening structure coalesce, and form one or more large abscesses; these abscesses either terminate fatally or open into the pleural cavity, causing *empyema* and exhaustion, or open into the bronchi and are expectorated, or an *interstitial pneumonia* is developed and the abscess encapsulated in a firm cicatricial tissue.

Gangrene of the lungs may result from blocking up of the bronchial or pulmonary arteries by coagula during any stage of the disease.

The uninflamed portions of the lungs are hyperemic and their functional activity is increased.

Death sometimes results from a general edema of the unaffected lung, such cases being often erroneously termed *"double pneumonia."*

If inflammation of the pleura be associated with a pneumonia, the so-called *pleuro-pneumonia*, the changes in the pulmonary pleura are characteristic. "An uneven, thin, downy-looking layer of plastic exudation covers its surface. This plastic layer may conceal the liver-brown color of the pneumonic lung. As the third stage is reached, the opposing surfaces of the pleura may become agglutinated. The pleuritic changes follow very closely those which occur within the lung. The cells in the pleuritic exudation are mainly pus. The pleuritic membrane is opaque, congested, and ecchymotic. It may become so thick as to give a dull note on percussion, after resolution is reached."

Duration of Stages: stage of congestion, from one to three days; *stage of exudation,* from three to seven days; *stage of resolution,* from one to three weeks.

In severe cases, or in the very young, the aged, or the depressed, the stage of red hepatization may be fully developed within forty-eight hours.

Endocarditis, either simple or malignant, is a common accompaniment. Pericarditis is frequent. The spleen is usually enlarged and soft.

Symptoms.—The affection begins with a severe and usually protracted chill (in children often convulsions, and in adults sometimes vomiting), followed by a rapid rise of temperature, 103°-104° F., a strong, full, but rapid pulse, soon showing evidence of embarrassed cardiac action from obstruction of the pulmonary circulation. There are also present, either a dull or sharp pain near the nipple, aggravated by pressure, breathing, or coughing; shortness of breath, the inspiration short and superficial, the expiration accompanied by a moan or grunt, the number of respirations increasing to 40, 50, or more a minute, causing interrupted speech; disturbance of the ratio between pulse and respiration; and cough, at first short, ringing, and harsh, followed by a scanty, frothy, mucoid expectoration. The sputum soon becomes transparent, viscid, and tenacious, changing about the second day to the familiar rusty sputum. The quantity is increased and a yellow color is assumed as the disease advances. In rare instances, cases occur in which the bloody or blood-streaked sputum continues throughout the disease. Microscopic examination of the sputum in simple cases shows it to contain red blood cells, blood pigment, pus cells, the characteristic diplococci, and various other microörganisms.

From the very onset of the disease, the prostration is of the most pronounced character. The countenance is flushed, and especially over the malar bones there is a well-defined mahogany blush. The lips are more or less blue and herpes may be observed upon them. Epistaxis, headache, sleeplessness, and gastric disturbances are common. The tongue is coated, the appetite is impaired, and there is constipation. Delirium is sometimes present, and when occurring early is a grave sign. The urine is small in amount, highly-colored, deficient in chlorids, and often slightly albuminous. The blood shows evidences of leukocytosis.

The fever usually reaches its maximum within twenty-four hours and continues high, with diurnal remissions, until either the fifth, seventh, ninth, or eleventh day, when a crisis occurs, and within twenty-four hours all the symptoms are decidedly lessened, the fever absent, and convalescence is established, followed by rapid recovery. Occasionally, the termination is by lysis.

Physical Signs.—*Inspection* reveals during the first stage deficient movement of the affected side, due to pain. The apex-beat is normal in situation, and the interspaces do not bulge. In the second stage the healthy side rises normally, the affected side lagging behind. If both lower lobes are impervious to air, the diaphragm cannot descend and the epigastrium does not project during inspiration, the breathing being conducted by the upper part of the chest (superior costal respiration).

Palpation during the first stage shows the vocal fremitus to be more distinct than normal, especially over the diseased portions. In the second stage, the vocal fremitus is markedly exaggerated, except in those rare instances of occlusion of the bronchi by secretion. The cardiac impulse is felt in the normal position.

Percussion.—In the first stage, the percussion note is slightly impaired at times, having a hollow or tympanitic quality. In the second stage, there is dullness over the affected parts, with an increased sense of resistance. Over unaffected adjoining areas, the resonance is increased (Skoda's resonance).

Auscultation.—In the first stage there is heard over the affected part, a feeble vesicular murmur, associated with the true vesicular or crepitant (cracking) râle, most distinct during inspiration. In the second stage there is harsh, high-pitched, bronchial respiration, at times resembling a to-and-fro metallic sound, except in those rare instances in which the bronchi are more or less filled with secretion. Bronchophony, or distinctly transmitted voice, is present and at times pectoriloquy, or distinct transmission of articulated sounds, may be heard. In the third stage, the breathing changes from bronchial to broncho-vesicular and the crepitant râle (crepitatio redux) returns. As resolution proceeds, the breath sounds are associated with large and small moist and bubbling râles. According to Da Costa, the physical signs, symptoms, and morbid phenomena of this malady correspond usually in the following manner:

I. Stage of engorgement and beginning exudation.	Crepitant râle; slight percussion dullness.	Cough; beginning dyspnea and rapidly developed fever heat.
II. Stage of solidification of lung tissue (red hepatization).	Percussion dullness; bronchial respiration; bronchophony.	Rusty-colored sputum; dypnea; cough; high fever with marked evening exacerbations and morning remissions.
III. Stage of softening (gray hepatization.)	The same physical signs as in the second stage, unless large abscesses have formed.	Chills; prostration, etc.; purulent or brownish sputum; generally high temperature.

Clinical Varieties.—*Typhoid pneumonia* is a term applied to those cases which are accompanied by signs of extreme prostration, delirium, tremor, very high temperature, and profuse and prolonged exudation. They may also terminate by a crisis.

Bilious pneumonia occurs in cases accompanied by congestion of the liver or bile ducts; the result of venous stasis from pulmonary obstruction or from an accompanying acute catarrhal jaundice. In malarial districts pneumonia and malaria are often associated, when jaundice more or less pronounced occurs. Such cases are termed malarial or intermittent pneumonia.

Alcoholic, or pneumonia of the intemperate, has one very characteristic symptom, to wit.: early delirium. In pneumonia generally the mind is clear, even when all the conditions are unfavorable. Pneumonia of the intemperate may begin with symptoms closely resembling an attack of delirium tremens, cough, expectoration, and pain being very slight, or even absent.

If *purulent infiltration* follow the stage of red hepatization, instead of the crisis, symptoms of exhaustion occur, with profuse purulent expectoration, high temperature, severe sweats, the tongue brown and dry, sordes collecting on the gums, low delirium, feeble pulse, rapid, rattling breathing, the recovery slow, and convalescence tedious.

Pneumonia in the aged or the insane may be latent, coming on without chill or pain and with only a slight fever; the cough and expectoration are slight, physical signs ill-defined and changeable, and the constitutional symptoms out of all proportion to the amount of lung involved.

Apyretic pneumonia is that which lacks fever, and is the result of exhaustion and the depressing effect of the infecting agent on the nervous system. They may occur as the result of embolism.

Aspiration pneumonia is due to the aspiration of fluids of any kind, the disease being really of mechanical origin.

Traumatic pneumonia is the variety resulting from severe contusions of the chest, the trauma predisposing to the disease by mechanical injury of the lung, the diplococcus finding suitable nidus at the site of injury.

Pneumonia in Children is marked by nervous phenomena. Convulsions often usher in the attack, and headache, delirium, coma, and hyperpyrexia are prominent symptoms.

Terminations.—Asthenic cases recover within two weeks. When purulent infiltration supervenes, the disease pursues a tedious course of several weeks' duration, with a low exhaustive fever.

If death occur during the first or second stages, it is usually the result of a collateral edema of the uninflamed lung, profound toxemia, or cardiac failure and impaired nerve-force.

If *abscesses* occur, there are exhausting sweats, frequent cough, with a large amount of yellowish-gray, at times blood-streaked, expectoration.

Gangrene of the lungs is a rare termination; it is associated with symtoms of collapse, the expectoration of a blackish, fetid character, with the physical signs of a pulmonary cavity.

Fibroid induration or pulmonic cirrhosis and phthisis are occasional terminations.

Complications.—*Acute pleuritis* is a frequent complication of croupous pneumonia, occurring as often as from ten to twenty-five percent of cases. The more acute localized pain, the greater embarrassment of respiration, and the usual physical signs of effusion are the evidences of a *pleuro-pneumonia.*

Endocarditis is a common complication, showing irregular but protracted temperature record, with chills and sweats and great embarrassment of the respiration.

Capillary bronchitis is a rare but dangerous complication. *Pericarditis, rheumatism,* and *gout* are rare complications.

Diagnosis.—*Edema of the lungs* may be confounded with the first stage of pneumonia, but the subsequent history, its presence on both sides, and the waterish expectoration and absence of chill and pain and the physical signs of pneumonia soon determine the diagnosis.

Pleurisy is more often confounded with pneumonia than any other disease, the points of distinction between which will be pointed out when discussing that affection.

Typhoid fever, when accompanied by hypostatic congestion of the lungs, may be mistaken for pneumonia; the history, mode of onset, temperature record, etc., will aid in making the correct diagnosis. Hypostatic congestion occurs late in typhoid, while pulmonary congestion is the earliest manifestation in pneumonia.

Acute Phthisis may resemble the affection closely, and is only differentiated with certainty by finding the tubercle bacillus in the sputum.

In Bright's disease, valvular heart disease, diabetes, and alcoholism, the condition of the lungs should be ascertained at frequent intervals, as these affections are prone to be complicated with pneumonia.

Prognosis.—This depends largely upon the extent of the inflammation. Double pneumonia is especially grave. The disease is uncertain; the mortality ranging from 20 to 40 percent. In young adults of temperate habits, the outlook is, as a rule, good, while in the aged and intemperate the prognosis is bad. Pneumonia in drunkards almost invariably terminates fatally. Typhoid pneumonia, pneumonia in the insane, bilious pneumonia so-called, purulent infiltration, abscesses of the lung, and gangrene, all have a grave outlook. Heart or kidney disease influence pneumonia unfavorably. Cases in which the temperature is subnormal or is extremely high are also very grave and seldom recover. A very rapid pulse, severe nervous symptoms, and the absence of leukocytosis are unfavorable symptoms. Meningitis in the course of pneumonia usually terminates fatally.

Treatment.—If pneumonia be regarded as a constitutional malady with a local lesion, then the consolidated lung no more calls for treatment than does the intestinal ulcer of typhoid fever, and the *general* condition of the patient is to govern in the management and not the local changes in the thorax. A simple pneumonia attacking persons previously in good health requires no more active treatment than any of the so-called self-limited diseases, provided only that the extent of the disease be moderate, and there be no complication.

The patient should be placed at rest in bed in a moderately heated and well-ventilated room, and protected from all draughts.

The diet must be of the most nutritious but easily digestible character, and given at periods of every three hours, watching that the food is assimilated. A distended stomach and abdomen are dangerous. Strong black coffee throughout the disease is valuable. Liquid or semisolid substances, such as milk, broths, eggs, etc., are especially serviceable. A cotton jacket should be applied to the chest, unless contraindicated by other local treatment.

The much-discussed question of venesection is now a settled problem in the affection; if we bleed, it is "*not because of pneumonia, but in spite of pneumonia.*" Called to a patient in the first stage or early in the second stage, who has been vigorous and otherwise healthy, with a high temperature, 105° or more, with frequent pulse, one hundred and twenty beats or more, or a slow, full pulse showing cardiac oppression, flushed surface, and marked dyspnea, a copious bleeding is indicated, and the same may be said when symptoms of collateral edema threaten; this is bleeding for symptoms and not for the disease *per se.*

There is no remedy which can exert a favorable influence upon the pneumonic process. Many cases recover without, and many cases in spite of, treatment. When treatment is instituted, be guided by the fact that you are not to treat pneumonia, but a patient with a pneumonia.

At the onset, if venesection is not indicated, relief of the pain may follow the use of dry or wet cups. If the tongue be coated and the gastrointestinal canal deranged, a calomel purge is indicated.

℞. Hydrargyri chloridi mitis	gr. ij	.13 Gm.
Sodii bicarb.	gr. iv	.26 Gm.
Pulv. ipecac.	gr. j	.065 Gm.

M.—Ft. chart. No. iv.

S.—One every two hours, followed in two hours after last powder by mild saline.

Action on the skin and kidneys by refrigerant mixtures or small doses of Dover's powder is valuable. The administration of the arterial sedatives, aconite and veratrum viride, is recommended by Drs. Da Costa and H. C. Wood. In pneumonia of children, the use of small, frequently repeated doses of tincture of aconite, in the early stage, is most useful.

Poultices are of slight value, but home-made mustard plasters, weakened with flour, may be used in all stages. If the heart be weak from the onset, either of the following are valuable: digitalis, caffein citrate, nitroglycerin, spartein, or strychnin. Indeed, it seems a good practice to administer strychnin in full doses from the onset. Quinin sulphate, gr. ij-v (0.13-0.3 Gm.) every three or four hours, is always valuable.

Second Stage.—During this period the indications are to maintain the heart's action and to lessen the fever. Cardiac failure being one of the most common caûses of death in pneumonia, it is highly important to sustain the heart from the very beginning. Strychnin sulphate, gr. 1-32 to 1-20 (0.002-0.003 Gm.), administered every three hours, by the mouth or hypodermically, caffein citrate, gr. ii to v (0.13-0.3 Gm.) every four hours, or tincture of strophanthus, ♏v-x (0.3-0.6 Cc.) every three hours, are valuable cardiac tonics in pneumonia. The employment of digitalis and nitroglycerin depends upon the condition of the pulse. If the tension is low, the result of relaxation of the peripheral blood-vessels —vasomotor paralysis—digitalis in full doses is indicated; but if the tension is high, with embarrassed right heart, nitroglycerin, combined with aromatic spirits of ammonia, should be administered every hour or two. Alcoholic stimulants judiciously employed are most efficient means for preventing or overcoming the cardiac failure. The amount can only be determined by a careful study of each case, as a few ounces in the twenty-four hours may answer in one, while another may require eight or ten ounces. It is well to begin with small doses, increasing or decreasing as its effects are good or bad. *The indicator of the heart's condition is the pulse.* In the aged, the feeble, or in those accustomed to the use of alcohol, stimulation is indicated from the onset. Other indications would be a frequent, feeble, irregular, or intermitting pulse; a dicrotic pulse; delirium, muscular tremor, and subsultus; immediately following crisis, and the period of collapse. The hypodermic injection of normal salt solution is also recommended as a valuable tonic and stimulant. When collapse threatens, camphorated oil, hypodermically, is of great service.

Reduction of temperature is very necessary in many cases. If the fever is under 103° F., cool sponging with alcohol and water, or water alone, is sufficient. If the temperature is above 104° F., antifebrin, gr. v (0.3 Gm.) may be used every three hours until a reduction occurs. Strychnin sulphate, or caffein citrate, may be added to each dose. Phenacetin or acetanilid are also valuable, and considered less depressing, but it is to be remembered that a temperature under 104° is as normal to pneumonia as the dyspnea or the rusty sputum, and consequently antipyretic drugs should be used with caution. The use of the cold pack or of cold baths for reducing the temperature in acute pneumonia has not given the success expected.

Dr. Mays strongly advocates the use of ice-bags to the chest in pneumonia. He says: "Very often it is found that the application of the ice to an affected spot is immediately followed by a marked lowering of the temperature and improvement in the physicl signs in the part." I am able, from a large experience, to endorse this statement.

For dyspnea and pain the cardiac stimulants should be continued, and in addition morphin sulphate should be administered hypodermically as the occasion requires. Counterirritation to the chest will also relieve the pain. The inhalation of oxygen will lessen the shortness of breath, but too much should not be expected from this remedy, as there is some additional factor besides the mechanical one of consolidation of the lung producing the dyspnea, for the consolidation is just as marked immediately after the crisis, while the dyspnea is wonderfully relieved.

While the forms of treatment already given will in great measure lessen the cough, there are times when something additional is necessary to relieve this distressing symptom. In such cases, morphin, codein, heroin, Dover's powder, citrate of potassium, ammonium chlorid, and ammonium carbonate, are especially beneficial.

Sleeplessness and delirium are best overcome by sulphonal, trional, chloralamid, chloral, or the bromids. The combination of strychnin and trional is of value. Opium is sometimes necessary, but is contraindicated in the presence of pulmonary edema or dyspnea.

Third Stage.—The treatment is a continuation of that of the second stage, with the addition of the following valuable combination:

℞.	Ammonii chloridi	......gr. v-x	.3-.6	Gm.
	Strychninæ sulph.	......gr. 1-24	.003	Gm.
	Aquæ chloroformi	.....fʒ j	4.	Cc.
	Syr. prun. virg.	.......fʒ iij	12.	Cc.

M. S.—Every three hours, diluted.

In all cases, the condition of the heart should be carefully watched and the medication and dosage should be guided largely by its action. This is particularly true in the asthenic varieties of the disease.

During convalescence, the diet should be highly nutritious, and iron, quinin, strychnin, wine, malt liquors, cod-liver oil, etc., should be administered. If consolidation shows any tendency to linger, blisters should be applied locally and the iodids administered internally.

The serum treatment and the antiseptic treatment, so-called, are as yet not generally accepted and are still under consideration.

CATARRHAL PNEUMONIA.

Synonyms.—Broncho-pneumonia; lobular pneumonia; capillary bronchitis(?).

Definition.—An acute catarrhal inflammation of the bronchioles and alveoli of the lungs, characterized by fever, cough, dyspnea, copious expectoration, and great depression.

Causes.—It may be due to an extension downward of a bronchial catarrh, or it may follow one of the infectious fevers, especially measles, influenza, and whooping cough. Persons of the rachitic or scrofulous diathesis, in whom there is a greater irritability of the epithelial elements, are particularly predisposed to this form of pneumonia on slight exposure. It may also be due to influenza or heart-disease. The affection is observed most frequently in childhood and old age. The inspiration of particles of food and mucus, such as occurs in palsies, uremia, last stages of low diseases, etc., induces pneumonia of the catarrhal type (aspiration or deglutition pneumonia).

The exciting cause is a microörganism or group of microörganisms. Mixed infection is the common condition. The organisms found with greatest frequency are the micrococcus lanceolatus, the streptococcus pyogenes, the staphylococcus aureus and albus, and Friedländer's bacillus. In some cases, the colon bacillus, the typhoid bacillus, Kleb's-Löffler bacillus, or the bacillus of pneumonia may be demonstrated.

Pathologic Anatomy.—The earliest change is hyperemia of the mucous membrane of the bronchi, extending to the connective-tissue of the bronchioles and accompanying arterioles and to the alveoli, with swelling and succulence of these tissues, accompanied by an abnormal secretion and an immense production of young cells from the proliferation of the bronchial and alveolar epithelium, admixed with a yellowish, creamy, mucoid material, which blocks up the bronchioles and air cells.

Both lungs are involved, and on section scattered areas of consolidation are observed surrounding the finer bronchioles. Collapsed areas may be noticed in addition, due to obstruction of the bronchi. The terminal bronchi are found filled with a purulent exudate. This exudate and the infiltrate in the lung-tissue are made up of desquamated epithelium and leukocytes.

The affected parts first have a reddish-gray, soon changing to a yellowish-gray, color, due to the rapid metamorphosis of the newly developed cells. If the fatty change be completed, absorption takes place and the consolidation is removed; if it remain incomplete, the cells atrophy, the little mass becoming caseous, and the disease passes into a chronic state.

The bronchial tubes also participate in the disease; the walls become thickened, from a hyperplasia of the connective-tissue (*peribronchitis*), and their caliber is often increased.

Symptoms.—Catarrhal pneumonia begins as a catarrhal bronchitis. It may be either acute, subacute, or chronic in its course.

Acute variety: Its onset is announced by a gradual rise of temperature to 102°-103° F., the febrile phenomena assuming a typical remittent character, with rapid, laborious, and shallow breathing, as shown by the widely dilated nares and violent action of all the accessory muscles, while the insufficient distention of the lungs is shown by the great recession of the lower part of the chest-walls and sinking in of the intercostal spaces. The inspiration is short and imperfect, the expiration noisy and prolonged; the pulse is frequent, 100-120 or more, and somewhat compressible; the cough, which, during bronchitis, was loose, now becomes short, hacking, dry, and painful, soon followed by more or less copious mucopurulent expectoration; the appetite is impaired, the bowels somewhat loose, the urine scanty, high-colored, and the surface frequently covered with a more or less profuse perspiration.

The *subacute* and *chronic* varieties have the same general symptoms, but the duration is longer and the exhaustion greater.

The progress of catarrhal pneumonia is sometimes, although not often, a very acute one. The disease may prove fatal in a few days, especially if it attack feeble children; in such cases the countenance becomes pale and livid, the lips bluish, the eyes dull, and a restlessness supervenes, giving place to apathy, and a continually augmented somnolence.

Resolution, when it occurs, is by lysis, several weeks elapsing before complete recovery.

Physical Signs.—*Percussion* yields dullness in scattered areas over both lungs, the intervening healthy lung often giving a more or less hollow or tympanitic note.

Auscultation reveals vesiculo-bronchial breathing, changing to moist bronchial breathing, associated with small bubbling (subcrepitant) râles. As the disease progresses toward resolution, the râles become larger (larger bubbling) and more numerous. If pneumonic phthisis result, physical signs indicative of that condition are soon evident.

Sequelæ.—Attacks of catarrhal pneumonia complicated with atelectasis, or collapse of the lobules, when recovery occurs, are followed by emphysema of the lungs.

If the catarrhal products which fill the alveoli and bronchioles and intervening connective-tissue do not rapidly undergo complete fatty metamorphosis and consequent absorption, pneumonic phthisis results.

Diagnosis.—*Ordinary bronchial catarrh* differs from catarrhal pneumonia by the absence of dyspnea, fever, and dullness on percussion, and the presence of the large bubbling râles, and also by the subsequent history of the two affections.

Croupous pneumonia is a unilateral disease; catarrhal pneumonia is bilateral and diffused over both lungs—the former a self-limited disease, the latter having no fixed duration. Croupous pneumonia is characterized by acute onset, high fever terminating usually by crisis within ten days, and distinct physical signs, indicating uniform consolidation.

Acute tuberculosis at its onset is characterized by the presence of a capillary bronchitis, a differentiation being possible only by a study of the clinical history and course of the two maladies, and the presence of the tubercle bacilli in the former.

Edema of the lungs is a bilateral disease associated with a short, dry cough, and dyspnea, but lacks the previous catarrhal history and high temperature of catarrhal pneumonia.

Prognosis.—Fully one-half of the cases of true catarrhal pneumonia terminate fatally. The prognosis must be guarded in scrofulous or rachitic subjects, or those enfeebled by other diseases, for, unless prompt resolution can be effected, it will terminate fatally early, or develop pneumonic phthisis. I have seen cases continuing up and down for eight and ten months, and finally make a good recovery.

Treatment.—Confinement to bed is paramount, but the position of the patient is to be frequently changed. The diet must be of the most nutritious character, administered at frequent intervals: milk, eggs, chicken, beef, mutton and oyster broths are the most suitable articles. The steady use of brandy or whiskey throughout the attack is of importance, regulating the amount by the age of the patient and the severity of the attack.

Locally, a weak mustard plaster followed with a cotton-batting jacket is valuable. Poultices are of little use. The febrile symptoms and early cough are often modified by the following mixture:

℞.	Potassii citratis	ʒ vj	24. Gm.
	Spts. ætheris nitrosi	fʒ iv	15. Cc.
	Tinct. opii camphorat. ..	fʒ iv	15. Cc.
	Liquor. potassii citratis	ad f℥ vj	ad 180. Cc.

M. S.—Dessertspoonful every three hours.

Early in an attack, with high temperature in children, tincture of aconite, in small, frequently repeated doses is valuable. If the fever persists, a combination of phenacetin or antifebrin with camphor or digitalis is useful. The ice-bags or poultices are as strongly recommended for broncho-pneumonia as for croupous pneumonia, and in sthenic cases should be given a trial.

For the catarrhal process, the air of the apartment should be maintained at an even temperature and moistened by disengaging the vapor of water in it. The following combination is of great utility in nearly all cases, regulating the dose in accordance with the age of the patient:

℞.	Ammonii carbonat. .....gr. v	.3 Gm.
	Ammonii iodidi ....gr. v-x	.3-.6 Gm.
	Mucil. acaciæ ....q. s.	q. s.
	Syr. glycyrrh. ....fʒ j	4. Cc.
	Syr. prun. virg. q. s. ad fʒ iv	ad 15. Cc.

M. S.—Every three hours.

A more pleasant way of administering the ammonia salts is in capsules, each containing about two and one-half grains of each salt with an aromatic oil. Terpin hydrate acts remarkably well in many lingering cases. The aromatic spirit of ammonia in either chloroform or cherry-laurel water makes an excellent mild, stimulating expectorant.

During convalescence, tonics such as iron, cod-liver oil, syrup of the iodid of iron, etc., and good food are indicated.

FIBROID PNEUMONIA.

Fibroid pneumonia is a chronic disease of the lungs, characterized by a marked overgrowth of connective-tissue, or cirrhosis. It may, in rare instances, follow croupous or catarrhal pneumonia and chronic pleurisy. Inhalation of irritant particles of dust, stone, coal, etc., over a long period, are common causes. It is in most cases due to tuberculosis, but also arises independent of that affection. The signs, symptoms, and morbid anatomy of fibroid pneumonia and fibroid phthisis are the same, with the exception that the tubercle bacillus may be demonstrated in the sputum of the latter. See Fibroid Phthisis.

PULMONARY TUBERCULOSIS.

Synonyms.—Phthisis pulmonalis; phthisis; consumption; pneumonic phthisis; tubercular phthisis.

Definition.—An infective disease, caused by the *Bacillus tuberculosis,* the lesions of which are characterized by nodular bodies called tubercles or diffused infiltrations of tuberculous tissue, which undergo caseation or sclerosis, and may finally ulcerate, or, in some situations, calcify. (Osler.)

Clinical Varieties.—I. Acute miliary tuberculosis; II. Pneumonic phthisis; III. Tubercular phthisis; IV. Fibroid phthisis.

Cause.—It is now generally accepted that all varieties of pulmonary consumption are due to the active presence of the *Bacillus tuberculosis,* discovered by Koch in 1880. The lung-tissue must be in a receptive state, as the bacilli may be present in the respiratory tract without the development of the disease.

Any condition that lowers the tone of the general system renders the tissues susceptible to the changes produced by the tubercle bacilli. These will be enumerated in speaking of the clinical varieties of the disease.

ACUTE MILIARY TUBERCULOSIS.

Syonyms.—Acute phthisis; galloping consumption.

Definition.—An acute infectious *febrile* affection, due to the rapid eruption in various parts of the body, but especially in the lungs, of miliary tubercles; characterized by high fever, rapid pulse, hurried respiration, pains in the chest, cough, profuse expectoration, and rapid prostration.

Causes.—In the majority of cases it is the result of an auto-infection, arising from either an active or latent tuberculous focus. Cases develop in which no cause can be assigned. It often follows measles, whooping cough, variola, and influenza. The disease is most frequent between puberty and middle life.

"That the gray granulation be deposited throughout the body under the influence of certain conditions of irritation, it is necessary that a peculiar vulnerability of the constitution exist — in other words, that it be of the scrofulous type."

Pathologic Anatomy. — "The gray granulation, or miliary tubercle consists of a fine reticulation of fibers, with a mass of epithelioid cells and granules, and often having a giant cell for its center." The deposit is generally over both lungs and the bronchial tubes, and is followed by hyperemia, increase of secretion, having a viscid and adhesive character, and the destruction of all the tissue with which it comes in contact.

Deposits also take place in the brain, pleura, intestines, peritoneum, and kidneys.

Clinical Forms.—*General or typhoid, pulmonary,* and *cerebral.*

Symptoms.—*The typhoid variety* of the disease or generalized miliary tuberculosis is characterized by gradual, progressive weakness, loss of appetite, dry, clean tongue, constipation, flushed cheeks, irregular fever, the temperature seldom going above 103° F. or 104° F., rapid, feeble pulse, and mild delirium. The respirations are increased and in the early stage cough and expectoration are slight. Frequently, symptoms of a diffused bronchial catarrh of the smaller tubes are present. Excessive sweating is common. As the disease progresses, the prostration becomes more profound, cyanosis develops, and delirium, stupor, coma, and finally death supervene.

Being a general infection of asthenic type, it is liable to be mistaken for typhoid fever. The chief points of difference are the absence of the typical typhoid or step-like fever record, roseolar eruption, diarrhea, Widal reaction, and diazo reaction in miliary tuberculosis. The presence of tubercle bacilli and tubercles in the retina and choroid are conclusive evidences of general tuberculosis. The possibility of malaria should be eliminated in all cases by examination of the blood for the plasmodium.

Acute general tuberculosis always progresses towards a fatal termination. The affection seldom lasts more than six or eight weeks, but may be prolonged for a greater period. The treatment is, therefore, necessarily unsatisfactory, and must aim merely to relieve distressing symptoms. Liquid or semisolid food, such as milk, eggs, broths, etc., and stimulants should be freely administered. Hydrotherapy should be used to control the fever and anodynes should be employed to lessen the cough and restlessness.

The *pulmonary variety* is characterized by sudden onset, with chill or chilliness, followed by fever, 102° F., to 104° F., rapid dicrotic pulse, 120 to 140 per minute, cough, with scanty, glairy expectoration, increased respiration, 30 to 60 per minute, pain in the chest, hot skin, dry tongue, and deranged digestion. Prostration is profound. As the affection advances, the symptoms increase in severity; cyanosis soon becomes manifest; the sputum becomes more abundant and often rusty in color; hemoptysis may occur; emaciation and anemia are marked, and later there supervene headache, vertigo, sleeplessness, delirium, coma, and death. Tubercle bacilli and elastic fibers may be found in the sputum, and an examination of the blood reveals an increase in the number of white cells (leukocytosis). When the tubercles are formed in the meninges or in the intestinal wall, symptoms referable to these structures are superadded.

The physical signs are not constant. The percussion resonance is normal until the deposits become considerable, when it is either slightly impaired or at times even tympanitic. With the development of cavities, the amphoric percussion-note may be obtained. On auscultation, often very little change may be detected in the vesicular murmur, but diffused râles of bronchial catarrh may be heard. In some cases, vesiculobronchial breathing, associated with large and small, moist or bubbling râles may be present, soon followed by bronchial and bronchocavernous breathing, with large and small circumscribed moist and bubbling râles.

This variety terminates in death in from four to twelve weeks. In rare instances, it may be of several months' duration. It may be mistaken for typhoid fever with marked pulmonary complications, but a careful history and examination of the blood, sputum, and urine will serve to make the proper diagnosis.

Treatment is of no avail in bringing about a cure. According to Loomis, morphin, gr. 1-20 (0.003 Gm.) hypodermically every six or eight hours, is of great benefit in staying the progress of the disease, prolonging life, and keeping the patient comfortable. Mc-Call Anderson states that subcutaneous injections of atropin sulphate check the exhausting sweats, and that quinin sulphate, digitalis, and opium reduce temperature. As an alternative to the latter procedures, he advises ice-cloths to the abdomen. Hydrotherapy is always of value in this connection. Free stimulation is always necessary and the various symptoms should be combatted as they arise.

The *cerebral variety* will be described in detail in the section on nervous diseases, to which the reader is referred.

PNEUMONIC PHTHISIS.

Synonyms.—Chronic catarrhal pneumonia; catarrhal phthisis; caseous pneumonia; caseous phthisis; phthisis florida.

Definition.—A form of pulmonary consumption characterized by the destruction of the pulmonary tissue resulting from the action of the bacillus tuberculosis, causing the caseation or cheesy degeneration of inflammatory products in the lungs, and the subsequent softening and destruction of the caseous matter; characterized by hectic fever, cough, shortness of breath, purulent expectoration, and more or less rapid prostration.

Causes.—In this as in other forms of tuberculosis, the tubercle bacillus is the primary cause. A condition of impaired health, such as results from unhygienic surroundings, exposure, or overwork, or such as accompanies the strumous diathesis or constitutional diseases is an important contributory cause. A catarrhal pneumonia in any portion of the lung, but especially at the apex, inflammation occurring around a blood clot, and the constant inhalation of irritant particles are also factors of great etiologic importance. In many instances the disease follows one of the infectious fevers.

Pathologic Anatomy.—The tubercular infiltration is at first peripheral and rapidly leads to active inflammation, which is manifested as a broncho-pneumonia, the bronchioles and air-vesicles being blocked with cheesy material. As a result, opaque white foci, 5 to 12 mm.in diameter, are disseminated throughout the lung,between which are congested but crepitating areas. The diseased foci tend to soften, rapidly resulting in small abscess cavities. The tuberculous areas may be widely separated, or may be limited to certain regions, especially the apices. The process, in rare instances, may be grafted upon a croupous pneumonia in which resolution has failed to occur. It may be distinguished from croupous pneumonia by the greater disintegration of tissue. When a pneumonia terminates in resolution, the inflammatory products are absorbed by first undergoing a fatty metamorphosis. If the fatty metamorphosis be incomplete, the cells are atrophied and undergo the caseous degeneration, which consists in the absorption of the watery parts, the fatty degeneration of the cellular elements, and the granular disintegration of the fibrinous material, so that ultimately a soft, solid mass is produced, yellowish in color, having a cheesy appearance.

The situation of the pneumonia resulting in the above changes is usually at the apex or under the lower inner scapular region, but it may occur at any portion of the lungs, or a whole lung becomes infiltrated, and undergoes the cheesy degeneration (phthisis florida). As in croupous pneumonia and other pneumonic conditions there is a great tendency towards involvement of the pleura.

Symptoms.—Pneumonic phthisis occurs in three clinical forms—acute, subacute, and chronic.

The *acute variety*, or *phthisis florida*, so-called, runs a very rapid course, beginning either as a croupous or catarrhal pneumonia involving an entire lung or portions of both lungs, and is accompanied by high, but variable, temperature, 103°-105° F., remittent in type, profuse night-sweats, shortness of breath, severe cough, profuse, purulent, and blood-streaked expectoration containing tubercle bacilli, anorexia, and feeble digestion. There is rapid loss of flesh and strength; the patient succumbing in a few weeks or months from exhaustion. A decided remission in the local and general symptoms in this form may take place, the disease afterwards pursuing a more chronic course.

In the *subacute variety* there is usually a history of an acute attack of pneumonia of one or two weeks' duration, which is followed by decided improvement, but not by complete recovery. After a lapse of some weeks or months pulmonary softening begins, destroying the lung-structure and ultimately leading to cavity formation. These changes are accompanied by chills, fever, night-sweats, emaciation, cough, and muco-purulent and blood-streaked expectoration containing tubercle bacilli. The affection terminates fatally within a year.

In the *chronic form* the origin is rather insidious, the patient having been susceptible to "colds" or "catarrhs" on the slightest exposure for an indefinite period. Cough appears, which gradually becomes persistent, with muco-purulent expectoration. Each severe cold is accompanied by chill, fever, pain in the chest, and either slight hemorrhages or blood-streaked expectoration. Finally the catarrhal symptoms become persistent and attended by morning chills, evening fever, profuse night-sweats, distressing cough, and profuse muco-purulent expectoration containing tubercle bacilli. Loss of appetite and feeble digestion are present, and weakness and exhaustion are profound. The symptoms continue to grow progressively worse, death occurring from exhaustion in from one to two years.

Physical Signs.—*Inspection* shows deficient respiratory movement over diseased portions of the lungs. The respiratory rate is increased.

Palpation over consolidated areas and cavities detects increased vocal fremitus.

Percussion reveals definite changes in lungs. The note at the apex varies from slight impairment of the normal note to dullness, and when cavities are formed there will be associated, scattered areas over which the tympanic or hollow note may be obtained. If the cavities communicate with a bronchial tube, the cracked-pot or cracked-metal sound is elicited. When the cavities are filled with exudation the percussion note will be dull, but after expulsion of the exudate, the tympanitic or cracked-pot sound may be again obtained.

Auscultation detects no impairment of the vesicular murmur in those portions of the lung free from disease; it is feeble or indistinct if many bronchioles are obstructed, and is harsh or blowing if the bronchioles are narrowed. After the lung has lost its elasticity, the inspiratory sound will be jerking and the expiratory sound prolonged and blowing in character. Associated with the impaired vesicular murmur is a fine, dry, crackling sound (crepitation), appearing at the end of inspiration. If bronchitis be associated, large and small moist or bubbling râles are also heard during respiration. When cavities form, either bronchial or broncho-cavernous respiration is heard, associated with more or less distinct gurgling râles. If the cavity be free from pus and have rather firm walls, the breathing is more amphoric in character.

Diagnosis.—*Catarrhal bronchitis* has many points of resemblance to pneumonic phthisis. The subsequent course of the latter, with the high temperature, prostration, emaciation, sputum containing bacilli, and physical signs will prevent error.

Acute fibrinous and *catarrhal pneumonia,* often after a course of two or three weeks, show the bacilli and yet are not recognized as tuberculosis. It is a safe rule of practice to suspect tuberculosis and examine daily for the bacilli in all cases of pneumonia that show the least tendency to linger, and particularly where there are chills and a remittent temperature record.

Prognosis.—*Acute phthisis* seldom lasts more than a few months, the *subacute* and *chronic varieties* may be prolonged for a year or two under good care and stimulating treatment.

TUBERCULAR PHTHISIS.

Synonyms. — Tuberculosis; consumption; incipient phthisis; chronic phthisis; chronic ulcerative phthisis.

Definition.—A chronic pulmonary disease caused by the bacillus tuberculosis, resulting in the deposition of tubercle in the lung structure, which in turn undergoes ulceration and softening, inducing septic infection, characterized by progressive failure of health, fever, cough, dyspnea, emaciation, and exhaustion.

Causes.—The direct cause is the tubercle bacillus. A susceptibility to its influence may be acquired by heredity, syphilis, alcoholism, chronic nephritis, occupations necessitating cramped postures, inhalation of foul air and irritating particles, etc., residence in dark, over-crowded, and damp apartments, catarrhal inflammation of the respiratory tract, and the infectious fevers. Debility from any cause, and early adult life are important predisposing factors. The infection usually takes place through the respiratory tract.

Pathologic Anatomy.—Careful examination of a lung affected with this form of tuberculosis will reveal a great variety of lesions. Among these may be mentioned nodular tubercles, diffuse infiltration, caseated masses, pneumonic areas, and cavities. Various changes may also be noted in the pleura, bronchi, and bronchial glands. The primary lesion is to be found usually from an inch to an inch and a half below the summit of the lung and near to the posterior and external borders (Fowler). From this region, the extension is downward. "A less common site corresponds on the chest-wall with the first and second interspaces below the outer third of the clavicle." The right apex is involved first in the majority of cases. Basic lesions are seldom primary.

The anatomical lesion, the tubercle, is of the same structure here as in other forms of tuberculosis. It first appears as a grayish-white, translucent, semisolid granulation, about the size of a millet-seed, usually deposited in the walls of the bronchioles or around the small blood-vessels. From its presence in these situations it induces a low form of inflammation which ultimately results in its destruction. The tubercles then undergo softening or cheesy necrosis with the formation of cavities and consequent destruction of lung-tissue. The small tubercles may coalesce, forming larger nodules, or diffuse tubercular infiltration.

The first effect of the tubercle bacillus is the formation of oval cells having a vesicular nucleus, due to proliferation of the fixed connective-tissue, endothelium, and epithelium (*epithelioid cells*). These constitute one of the characteristic features of the tubercle. In the center of this accumulation, may be found at a later period, a larger multinuclear mass *(the giant cell)*. Round or lymphoid cells are also present in abundance and may obscure the other cellular elements. New blood-vessels are never present in the tubercle, but the process may attack vessel-walls leading to their subsequent rupture and hemorrhage. The bacilli are to be found in the giant cells, between and in the epithelioid cells, and at a late period in the round cells. The cells soon become arranged concentrically, necrosis beginning in the center. The nodule may terminate in calcification, but more commonly the necrosis and liquefaction is unchecked and cavity formation is the result.

The method by which the tubercle bacillus may be detected has already been described in the introduction to the section on respiratory diseases *(q. v)*.

Symptoms.—The onset of the disease is very insidious and is attended by anorexia, dyspepsia, epigastric distress after meals, pallor, anemia, and weakness, all of which may serve to mislead the patient and physician. Later, there develops a slight, dry, hacking cough, referred to the throat or stomach and occurring usually in the morning, with scanty, glairy expectoration. As the deposition of the tuberculous disease progresses there occur irritable heart, gradual loss of weight, with impaired strength, more or less copious hemoptysis, and sharp pain, most marked below the clavicles. Slight "colds" serve to aggravate all of these manifestations.

The beginning of softening of the diseased area is marked by increased cough, with free expectoration containing tubercle bacilli and elastic tissue fibers, dyspnea increased on exertion, morning chills, evening fever, and night sweats (hectic fever), and diarrhea. The emaciation and weakness become profound, but the patient continues to be very hopeful.

With the formation of cavities, the cough becomes more aggravated. Expectoration is profuse and purulent; it may be greenish in color and made up of heavy coin-shaped plugs, which sink when placed in water (nummular sputum). Tubercle bacilli and yellow striæ are present. The pulse is rapid and weak. Hectic fever becomes more pronounced; the face is flushed; the eyes are bright; a sensation of burning of the soles and palms is present; and there are more copious night-sweats. Hemoptysis may occur at any time during the disease, but it is only during the later part of this period that the profuse hemorrhages are encountered. The blood in such cases is bright red, alkaline in reaction, and mixed with mucus. The emaciation, pallor, and weakness become extreme. Edema of the ankels occurs towards the end of the disease, indicating failure of the circulation. The mind remains clear and hopeful to the last.

Physical Signs.—*Inspection* during the early stage, shows slight depressions in the supraclavicular, and at times in the infraclavicular regions. While the configuration of the chest may be unchanged, it is more common to encounter the long, flat chest, with oblique ribs, prominent scapulæ, and deep depressions above and below the clavicles on either side (phthisical chest). As the disease advances, the emaciation, unilateral expansion, and localized retraction may be observed.

Palpation serves to detect increased vocal fremitus over either or both apices, and imperfect expansion.

Percussion yields a slightly impaired note in the early stage, at either or both apices. When the manifestations of the disease are prominent, dullness may be obtained over the consolidated areas. The regions in which it is most readily elicited are above and below the clavicles, in the supraspinous fossæ, and between the scapulæ. In the period of cavity-formation dullness may be detected with circumscribed areas of the amphoric, tympanitic, or cracked-pot sound. In order to obtain the cracked-pot sound over cavities, the patient should hold the mouth open and the chest should be struck quick and lightly.

Auscultation reveals, in the early stage, jerky inspiration with crackling râles at the apex, and prolonged, high pitched expiration. Later the breathing becomes distinctly harsh and is associated with subcrepitant, and large, moist or bubbling râles. There is increased vocal resonance. Coughing will always serve to render the râles audible. In the stage of cavity-formation bronchial, bronchovesicular, and cavernous or amphoric breathing are obtained, and variously sized bubbling or gurgling râles are heard. Bronchophony, and pectoriloquy may be elicited.

Complications.—The tuberculous process may simultaneously affect the brain and its membranes, nerves of special sense, larynx, pleura, intestines, peritoneum, ischiorectal cellular tissue, endocardium or pericardium, the symptoms of which are then superadded to those referable to the pulmonary condition. Amyloid degeneration of the viscera is a common complication.

Diagnosis.—The early diagnosis of phthisis rests largely on the history, the symptoms, especially the gastric disturbances, evening fever, and accelerated pulse, and the physical signs. The presence of the tubercle bacillus in the sputum is conclusive evidence of the disease. In all suspected cases, the chest and the expectoration should be carefully examined.

Prognosis.—Generally speaking the outlook is very unfavorable. The duration is usually about two years, death occurring from exhaustion. Many cases under the influence of good hygiene, sunshine, stimulating food, dry rarefied atmosphere, and appropriate treatment of every symptom, are prolonged a more or less indefinite period. Arrest of the disease, when it occurs, is due to calcification of the tubercles. The unfavorable symptoms are rapid pulse, high temperature, marked gastric disturbances, and manifestations due to tuberculosis elsewhere in the body.

FIBROID PHTHISIS.

Synonyms.—Chronic interstitial pneumonia; cirrhosis of the lungs; Corrigan's disease.

Definition.—A hyperplasia (thickening) of the pulmonary connective-tissue, resulting in atrophy and degeneration of the vesicular structure, associated with bronchial inflammation; characterized by cough, profuse expectoration containing the bacillus tuberculosis, fever, emaciation, and ultimately death by asthenia.

Causes.—The exciting cause is the tubercle bacillus but heredity, inhalation of irritants, such as occurs in the pursuit of occupations, such as stone-cutting, grinding, mining, etc., lobar pneumonia, chronic bronchitis, alcoholism, syphilis, and chronic nephritis should be remembered as important etiologic factors.

Pathologic Anatomy.—The characteristic anatomical feature of this disease is the marked development of fibrous tissue in addition to the tubercular process in the lung. Contraction of the fibrous tissue and shrinking of the affected lung result.

Symptoms.—The affection is extremely chronic, beginning as a bronchial catarrh, which is worse in winter and better in summer, extending over a long period. In the more advanced stages of this disease, the cough is more persistent and expectoration is more copious, being made up of a muco-purulent material containing tubercle bacilli. Later hectic fever, with night-sweats, develops, and dyspnea and rapid emaciation become manifest. Edema of the ankles is a late sign and depends on failure of the circulation. The termination is eventually in death.

Physical Signs.—Inspection reveals marked retraction of the affected side, due to shrinking of the diseased lung.

Percussion yields a dull note or impaired resonance, with scattered areas, over which hyper-resonance or tympany may be obtained.

Auscultation in the early stage serves to elicit vesiculo-bronchial or harsh respiration, associated with large and small moist or bubbling râles, but at a later period bronchial, broncho-cavernous, and cavernous breathing, with circumscribed gurgling râles, may be heard.

Diagnosis.—The distinctive features are the prolonged course, the bronchial catarrh worse during the winter, retraction of the lung, and the presence of the tubercle bacillus in the sputum.

Prognosis.—Death is the inevitable termination of this disease, but the course of the affection extends over a period from six to twelve years. Da Costa, in a study of one hundred cases of "grinder's consumption," found the average duration to be about twelve years from the development of the first symptoms.

TREATMENT OF PULMONARY TUBERCULOSIS.

Prophylactic Treatment.—The presence of the tubercle bacillus in the sputum, renders that substance a source of great danger since, after being dried, it is rapidly disseminated throughout the atmosphere. The sputum of all tuberculous patients should therefore be thoroughly disinfected by means of bichlorid of mercury solution (1 to 1000), carbolic acid solution (1 to 30), or caustic alkalies. Receptacles in which the sputum is collected, should contain a small quantity of water to prevent evaporation, and should be scalded in cleaning. Paper napkins, pasteboard spit-cups, rags, and similar inexpensive materials may be used to receive the expectoration and possess the great advantage that they may be destroyed completely by heat. Spitting upon the floor or in places other than the especially-provided receptacles should be prohibited. Excessive drapery and superfluous upholstery that do not permit of being easily cleaned, should be removed from the apartments of the tuberculous patient. The possibility of infection by means of milk and meat should always be considered. Infected meat and milk should be rejected as food, but an additional safeguard will be to thoroughly cook all meat and boil all suspected milk. Residence in low, damp, shaded localities should be avoided by those individuals predisposed to the disease — fresh air, sunshine, and out-door exercise should be advised. A high altitude where the air is dry and rare, and the climate equable, is of great advantage to such persons. Bathing and cold sponging, wholesome diet, and moderation in eating and drinking, should be prescribed. Localized foci of the disease should receive prompt attention.

Climatic Treatment.—Circumstances permitting, all patients in whom tuberculosis is detected, should be sent to a suitable climate. Those of a robust type are benefitted by high altitude and cold, and a certain degree of hardship or "roughing it." The change should be gradual. Disturbance of the circulatory system, cardiac weakness, small size of the heart, neurotic temperament, and persistent, high temperature contraindicate such a change in climate. Those who require protection should seek a residence in warm or equable and comparatively dry places at the sea-level or but slightly elevated In early cases, attended by persistent, high temperature, a sea voyage may be of great benefit. Colorado and New Mexico represent the high and dry climates, while Southern California may be taken as types of the warm and dry climates; warm and moist climates are encountered on the coast of Southern California and in Florida. When for any reason it is impossible to move the patient any distance, much can be done by placing the individual in some nearby country place, preferably among the hills and away from damp regions. Plenty of fresh air and sunshine may then be obtained. The city is no place for the consumptive, but the possibility of nostalgia and its deleterious effects should always be considered when ordering a change.

Hygienic Treatment.—The apartment in which the patient spends the greater portion of the day should be free from dampness and so situated as to be accessible to sunlight for as many hours as possible. The atmosphere should not be too dry, as cough and subsequent hemorrhage may be induced thereby. The temperature should average 65° F. It is desirable that the bedroom be occupied only at night, and be well ventilated during the day. The clothing should be warm and loose, being changed with the seasons. Woolen or silk underwear should be worn throughout the year. Heavy, oppressive clothing should be avoided. Frequent changes are necessary for obvious reasons. Daily bathing, followed by friction, is recommended. Cold sea-bathing is harmful. Rest and exercise in varying degrees in combination are beneficial. Exhaustion should always be avoided. Robust individuals may partake of outdoor exercise with benefit, while weak and anemic patients require rest and passive movements. The presence of high fever in always an indication for rest. Exposure to sunlight is very beneficial. Sedentary occupations should be avoided. The patient should be constantly amused. Nutritious food, such as meats, poultry, game, oysters, fish, animal broths, milk, eggs, etc., is always indicated. The quantity should be liberal. Nothing, however, should be fried. Among the articles to be avoided may be mentioned pork, veal, hot bread, cakes, pies, pastry, sweetmeats, rich gravies, crabs, lobsters, etc. Water should be taken freely. Whiskey, cod-liver oil, and stomachics aid the building up of the system and should be given with the food. Great care should be exercised not to discomfort the patient by overfeeding.

Medicinal Treatment.—Medicines should be administered for their general constitutional effect and also to relieve distressing symptoms. Cure, when it is affected, is only brought about by improving the general health and restoring the tone and resistance of the body. To this end, cod-liver oil, hypophosphites, alcohol, arsenic, and strychnin should be administered freely. Plain cod-liver oil, combined with the hypophosphites of calcium, sodium, and potassium (U. S. P.) is an excellent preparation, and when given with whiskey, is not distasteful. A tablespoonful twice daily is as large a dose as can be employed without disturbing the stomach. Should this occur, or as is usually the case, if there is indigestion from other causes, nux vomica, gentian, or other stomachics should be given. The following is very beneficial in this connection:

℞. Strychninæ sulph. ......gr. iv .26 Gm.
Aq. chloroformi, vel ....
Ess. pepsini ....f℥ ij 60. Cc.
M. S.—Ten minims equal gr. 1-24 (0.0025 Gm.).

Mode of administration: Five drops three times daily for one week, then ten drops three times daily for a week, then fifteen drops three times daily for a week, then twenty drops three times daily for a week, then fifteen drops, then ten drops, then five drops, and so on week after week for months.

Whiskey and brandy should be given in cases in which the asthenia is at all marked, the dose being guided by the effect produced and the exigences of the individual case. A rise of temperature and dyspepsia indicate withholding the alcohol. Arsenic is of great value also and may be given in the form of Fowler's solution or combined with digitalis.

℞. Acidi arseniosi ....gr. j .065 Gm.
Digitalini (Merck's) ...gr. j .065 Gm.
M. Ft. pil. No. xxx.
S.—One after meals.

Strychnin sulphate, gr. 1-60 to 1-30 (0.001-0.002 Gm.) after meals, and guaiacol, ♏iii-v (0.2-0.3 Cc.) for adults, and ♏ii-iii (0.12-0.2 Cc.) for children, four times daily, are of value. Creasote, gr. i (0.6 Gm.) after each meal, gradually increasing the dose, and creasotol, beginning with ten minims (0.66 Cc.) are also beneficial. The inhalation of modified air (pneumotherapy) and atmospheres saturated with the vapors of iodin creasote, carbolic acid, etc., has been employed with success. Counterirritation of the chest with blisters may be considered in obstinate cases.

Serum treatment may be accompanied by encouraging results in some cases. The refined tuberculin of Koch should be used and should be administered in doses short of that necessary to produce febrile reaction. The first dose should never exceed 0.2 mgm., and should be given hypodermically. The earlier it is employed, the more localized the disease, and the less general the infection, the greater will be the prospect of good results from its use. In cases in which fever and hemorrhage are present, it is contraindicated. The presence of mixed infection renders it useless. The results from its use in the wards of the Philadelphia Hospital were uniformly negative. It is not devoid of danger, and is of most value as a diagnostic agent.

The *cough* of pulmonary tuberculosis when slight may be readily relieved by the application of a mustard plaster, capsicum plaster, iodin, turpentine stupe, or a fly-blister to the chest over the region of most distress. Internally, creasote ♏i (0.06 Cc.) in milk or whiskey, three times daily, gradually increasing the dose, dilute hydrocyanic acid, ♏ii-iv (0.12-0.24 Cc.), terebene, ♏iii-x (0.18-0.62 Cc.), and similar expectorants should be administered. The various preparations of opium, especially paregoric, ʒi (3.6 Gm.), morphin, gr. 1-24 to ¼ (0.00275-0.0165 Gm.), codein, gr. ¼ (0.0165 Gm.), and heroin, gr. 1-20 (0.0033 Gm.), are of particular value in relieving this symptom.

℞.	Codeinæ sulphat. ......gr. ⅓-½	.022-.032	Gm.
	Acidi hydrocyanici dil. ..♏ ij	.12	Cc.
	Syr. tolu ....fʒ j	4.	Cc.

M. S.—One dose—to be given every three hours.

Or—

℞.	Codeinæ ....gr. iv	.26	Gm.
	Acid. hydrochlor. dil. ...fʒ ss	2.	Cc.
	Spirit. chloroformi ....fʒ iss	6.	Cc.
	Syr. limonis ....f℥ j	30.	Cc.
	Aq. lauro-cerasi ....ad f℥ iv	ad 120.	Cc.

M. S.—One teaspoonful, repeated when cough is troublesome.

Or—

℞.	Morphin. sulph.	gr. ss-ii	0.033-0.130 Gm.
	Potass. cyanid.	gr. iii	0.2 Gm.
	Acid. sulph. aromat.	fʒ i-ii	4. to 8. Cc.
	Syrup. prun. Virgin. q. s.	ad f℥ iii	90. Cc.

M. S.—Tablespoonful as often as necessary to quiet the cough (Tyson).

When coughing is harrassing or expectoration is difficult, as it often is in the morning, whiskey or a milk punch should be given in preference to any sedative.

In the pneumonic variety, the attempt should always be made to remove the caseous matter by absorption and expectoration. The following prescriptions will sometimes prove successful:

℞.	Ammonii carb.	gr. v	.3 Gm.
	Ammonii iodidi	gr. v-x	.3-.6 Gm.
	Aq. chloroformi	fʒ ij	8. Cc.
	Syr. prun. virg.	fʒ ij	8. Cc.

M. S. Every five hours, diluted,

Alternating with—

Liq. potassii arsenitis	♏ v	.3 Cc.
Mass. ferri carb.	gr. v	.3 Gm.
Vini xerici	fʒ j	4. Cc.
Aquæ	q. s. ad f℥ ss	15. Cc.

Dyspeptic symptoms are nearly always present, but may be aggravated by internal medication, in which cases the offending drugs should be lessened in dose or even temporarily suspended. These manifestations should be treated on general principles, the following formulas will often be found of benefit:

℞.	Pepsini cryst.	gr. ij	.13 Gm.
	Acidi hydrochlorici dil.	♏ xv	1. Cc.
	Glycerini	♏ xx	1.3 Cc.
	Succi limonis	♏ xv	1. Cc.
	Aquæ aurantii flor.	ad fʒ ij	ad 8. Cc.

M. S.—To be taken with meals, diluted; or:

℞.	Liquor. potassii arsenitis	♏ xxx	2. Cc.
	Tincturæ nucis vomicæ	fʒ j	4. Cc.
	Aquæ chloroformi	ad f℥ ij	ad 60. Cc.

M. S.—Teaspoonful before meals, diluted.

Fever may be materially lessened by rest alone, but in the event of its failing to do so, cool sponging or the use of phenacetin or antipyrin will be necessary. Niemeyer recommends the following:

℞. Quininæ sulph.	gr. j	.065 Gm.
Pulv. digitalis	gr. ss	.032 Gm.
Pulv. opii	gr. ¼	.016 Gm.
Pulv. ipecac.	gr. ¼	.016 Gm.

M.—Ft. pil. No. 1.
S.—One dose—to be taken as required.

The following is much more effectual:

℞. Quininæ sulph.	gr. x	.6 Gm.
Quininæ muriat.	gr. x	.6 Gm.
Pulv. opii et ipecac.	gr. iij	.2 Gm.

M.—Ft. capsul. No. ij.
S.—One capsule five hours and the other three hours before the expected rise of temperature.

Night-sweats are especially troublesome and may be relieved to a great extent by the administration, at bed-time, of atropin sulphate, gr. 1-100 to gr. 1-60 (0.00066 to 0.0011 Gm.), argaric acid, gr. ⅛-¼ (0.0082-0.0165 Gm.), camphoric acid, gr. xx to xxx (1.32 to 2 Gm.), picrotoxin, gr. 1-60 (0.0011 Gm.), or aromatic sulphuric acid, gtt xx (0.6 to 1.3 Cc.). Sponging of the body at bed-time with astringent solutions, such as the solution of alum and solution of white oak bark, or with alcohol, is beneficial. Tyson recommends a lotion of—balsam of Peru, 1 part; formic acid, 5 parts; chloral hydrate, 5 parts; trichloracetic acid, 1 part; absolute alcohol, 100 parts.

Diarrhea in the course of phthisis is greatly benefitted by the administration of bismuth subnitrate, gr. xx (1.3 Gm.), every three or four hours, with rest in bed and mustard to the abdomen. The following may be used:

℞. Cupri sulphat.	gr. iss	0.1 Gm.
Ext. nucis. vomicæ	gr. iii	0.2 Gm.
Pulv. opii	gr. vi	0.4 Gm.

M.—Ft. pil. No. xii.
S.—One every four hours.

Or—

℞. Liq. potass. arsenit. ..... ♏ xxx	2. Cc.
Tr. opii deodorat. ......fʒ iss	6. Cc.
Liq. pepsini.. q. s. ad f℥ ii	60.0 Cc.

M. S.—Teaspoonful at each meal.

Hemoptysis is best treated by absolute rest, with the application of an ice-bag to the chest and the administration of morphin, gr. ¼ (0.016 Gm.) hypodermically. Of almost equal value are atropin sulphate, gr. 1-200 to 1-60 (0.00032 to 0.001 Gm.), and spirits of glonoin (nitroglycerin), ♏i (0.06 Cc.). Gallic acid, salt, ergot, gelatin, and suprarenal extract are also recommended. If the hemorrhage is profuse the extremities should be circularly constricted by ligatures. The various measures usually advised for internal hemorrhage under other circumstances are applicable.

Pains in the chest are rather common. Strapping of the chest, mild counterirritation, or morphin hypodermically, will be required, according to their severity.

DISEASES OF THE PLEURA.

PLEURITIS.

Synonyms.—Pleurisy; "stitch in the side."

Definition.—A fibrinous inflammation of the pleura, either *acute, subacute,* or *chronic* in character, occurring either idiopathically or secondarily; characterized by a sharp pain in the side, a dry cough, dyspnea, and fever. It may be limited to a part, or may involve the whole of one or both pleural membranes.

Causes.—*Idiopathic pleuritis* is said to be due to cold and exposure, to injuries of the chest-walls, or muscular exertion. Tuberculosis is the cause in a few instances.

Secondary pleuritis occurs during an attack of pneumonia, pericarditis, rheumatism, variola, scarlatina, measles, Bright's disease, tuberculosis, or puerperal fever.

Chronic pleuritis follows an acute attack, or is the result of tuberculosis, Bright's disease, cancer, or alcoholism.

Pathology.—As in inflammation of other serous membranes there are five stages—hyperemia, exudation, effusion, absorption, and adhesions.

The first stage is marked by congestion and irregularly diffused redness of the membrane with scattered flakes of exudation. The second stage is characterized by the copious formation of lymph, which more or less completely covers the membrane, giving it a dull, cloudy, or shaggy appearance. If the inflammation ceases at this period, it is termed *dry pleurisy*. If the condition progresses an effusion is poured out into the pleural cavity, which may be sero-fibrinous, fibrinous, or purulent. The sero-fibrinous variety is most frequent and consists of a straw-colored fluid containing fibrinous flocculi, blood, and epithelial cells. Its quantity is usually rather large. When the exudate is fibrinous, the amount is small and the consistency is greater than that of the preceding. It undergoes organization quickly and gives rise to adhesions and pleural thickening. The exudate becomes purulent *(empyema)* only as the result of microörganismal infection. The effusion may become bloody in some instances as the result of ulceration (tuberculous or cancerous) and grave blood diseases. Displacement of the viscera is common in this stage; if on the right side, the effusion pushes the heart farther to the left; if on the left side, the heart is displaced to the right, the impulse often being seen to the right of the sternum. The lungs are also compressed and displaced upwards and against the spinal column. On removal of the fluid, they again expand, except in cases of chronic pleuritis, in which the adhesions interfere with the functional activity of the pulmonary structure. Absorption of the effused material is the natural sequence in most cases. Unabsorbed portions undergo organization, producing adhesions, which, in extreme instances may obliterated the entire pleural cavity. Sacculation of the effusion by adhesions is not uncommon, especially in purulent exudations in which the adhesions form an abscess wall. Varying degrees of adhesion of the opposing pleural surfaces are encountered, depending on the character of the exudate.

Chronic pleuritis results when the fluid is not absorbed or when it is effused into the cavity very slowly. The membrane is irregularly thickened and firm adhesions are formed within the meshes of which, the fluid is found. Retraction of the thoracic wall may be observed. If the fluid is pus, it may rarely become inspissated and encapsulated, or it may rupture through the chest-wall, discharging externally through a fistula, or it may rupture into the bronchi or in very rare instances into the intestines.

Symptoms.—The *acute variety* begins with a chill, followed by a sharp lancinating pain (stitch) near the nipple or in the axilla, aggravated by coughing and breathing, and associated with slight tenderness on pressure. The respirations are rapid and shallow, 30-35 per minute, and there are present a short, dry, hacking cough, moderate fever, and compressible pulse (90-120). With the effusion of liquid, the pain diminishes; dyspnea becomes more aggravated; cyanosis develops; the cough becomes distressing; and the cardiac action greatly embarrassed, the countenance wearing an anxious expression. The patient usually lies on the affected side. With the absorption of the fluid the symptoms gradually ameliorate, convalescence being rather rapid in simple cases.

The *subacute variety* begins insidiously after cold, exposure, and fatigue, in individuals enfeebled from various causes. The patients usually complain of a sense of weariness, shortness of breath, aggravated on exertion, evening fever, followed by night-sweats, and a short harrassing cough, with little or no expectoration. The pulse is small, feeble, but frequent, 100-120 beats per minute. The characteristic pain in the side, of acute pleurisy is absent.

The *chronic variety* is characterized by a more prolonged course, irregular chills, fever, night-sweats, dyspnea, palpitation, embarrassed circulation, and more or less prostration.

Physical Signs.—*Inspection* during the early stage serves to detect deficient movement of the affected side on account of the pain induced by full breathing. After the effusion has formed, there will be observed bulging or fullness of the affected side, with obliteration of the intercostal spaces and displacement of the cardiac impulse.

Palpation demonstrates feeble or absence of vocal fremitus over the effusion, with exaggeration of the same above the fluid. The affected side is immobile and very rarely fluctuation may be obtained.

Percussion during the early stage, yields a slightly impaired note. Later, dullness or even flatness, with increased resistance may be obtained directly over the fluid, while above the effusion the percussion-note is tympanitic. The line of demarkation is higher behind than in front. Effusion of the left pleural cavity obliterates Traube's semilunar space. The fluid changes its level with different positions of the body and the area of dullness is correspondingly movable.

Auscultation reveals during the early stage, a feeble vesicular murmur over the affected side, the patient breathing lightly to prevent pain. A friction sound, slight and grating or creaking, becoming louder as the exudation of lymph increases, limited usually to the angle of the scapula of the affected side, rarely over the entire side, accompanies the respiratory movements. During the stage of effusion, the vesicular murmur is feeble or absent on the affected side depending upon partial or complete compression of the lung by fluid. Above the effusion, puerile breathing is heard and just at the upper margin of the fluid a friction sound may be heard. Vocal resonance is diminished or absent over the fluid and markedly increased above the effusion, egophony being obtained at its upper margin. With the absorption of the fluid, the vesicular murmur and the moist friction sound gradually return.

Diagnosis.—*Acute pneumonia* may be distinguished from pleurisy by the pronounced chill, high fever, rusty sputum, increased tactile fremitus, bronchial breathing, fine, crackling, inspiratory râles, increased vocal resonance, fixed dullness, absence of intercostal bulging, and the absence of cardiac displacement.

Rheumatism of the intercostal muscles, or pleurodynia is characterized by more diffuse pain and tenderness. The physical signs are negative.

Enlargement of the liver may be mistaken for pleurisy with effusion, the chief point of distinction being that, in enlargement of the liver, the superior line of dullness is depressed upon full inspiration, while in pleurisy, with effusion, inspiration does not modify the location of the dullness.

Pericarditis with effusion is attended by physical signs limited to the precordium and symptoms referable to embarrassed circulation.

Empyema is attended by septic phenomena in addition to physical signs indicating pleural effusion. High and irregular fever, chills, sweats, and leukocytosis are present. Aspiration yields pus and pus-producing microörganisms.

Hydrothorax may be distinguished by its previous history, the absence of pain and fever, and on aspiration the withdrawal of an albuminous fluid of low specific gravity.

Prognosis.—Idiopathic pleurisy usually terminates in recovery within three weeks. Pleurisy, the result of constitutional causes has its progress modified by the condition with which it is associated. Empyema, unless the result of a diathesis, terminates favorably with prompt treatment. Double pleurisy is unfavorable. The etiologic factor of tuberculosis must always be borne in mind in making a prognosis in pleurisy, whether acute or chronic. When the effusion is very large the possibility of sudden death should always be considered.

Treatment.—The patient should be immediately placed at rest in bed and the diet restricted to liquids or semisolid substances. The administration of fractional doses of calomel, followed by a saline, should begin the medication. At the onset, in robust individuals, wet cups should be applied to the affected side to relieve the pain; if the pain is very severe, the dyspnea great, and the arterial tension high, venesection may be employed. In anemic and weak individuals dry cups should be used. Either wet or dry cups should be followed by the application of poultices or turpentine stupes. Severe pain is promptly relieved by the hypodermic injection of morphin sulphate over its site, repeated as necessary, or by the internal administration of small doses of Dover's powder. Strapping the affected side by means of broad strips of adhesive plaster is of benefit in all cases. In the very early stages, the disease may be cut short to some extent by the administration of sodium salicylate, gr. xv-xx (1-1.3 Gm.) well-diluted every three or four hours. The salicylates are also useful during the stage of effusion. After effusion has begun, fluid extract of pilocarpin, ♏xv (1 Cc.) every two or three hours, or in dram doses every other day for a week or two, and later twice weekly, or the following, should be administered:

℞.	Potassii acetat. ....gr. xxx	2.Gm.
	Infus. digitalis ....fʒ ij	8. Cc.

M. S.—Every three or four hours.

Matthew Hay, of Scotland, employs a concentrated solution of magnesium sulphate for the removal of the effusion. He advises that the patient should take nothing after the evening meal, and then an hour or so before breakfast, the salt (from ʒiv-vi (15-24 Gm.) to ℥i-ii (30-60 Gm.) dissolved in an ounce or two of water) is given, no fluids to be used after its administration. This usually produces from four to eight watery stools, without pain or discomfort, and also acts as a diuretic. Other diuretics, such as digitalis, caffein, potassium acetate, and Basham's mixture, may also be employed. Diaphoretics have little or no effect on the effusion. Bowditch advocates early aspiration. If after three or four days no impression is made upon the effusion by other means, aspiration should be employed, and followed by tablespoonful doses of Basham's mixture every four hours, and an early morning dose of magnesium sulphate, ℥ ss-i (15-30 Gm.) well-diluted. Perhaps a better plan would be, to be guided by the duration and character of the effusion, the degree of dyspnea and disturbance of the heart, and the visceral displacement. The puncture is usually made in the sixth or seventh intercostal space between the scapula and the axilla. If these means uninfluence the effusion, potassium iodid, gr. xv (1 Gm.), should be administered every four hours, diluted, and flying blisters should be applied over the affected side, or blue ointment (mercurial ointment) should be rubbed into the armpits, groins, and over the effusion. Painting of the affected side with iodin may also be employed.

In chronic pleurisy, blisters and iodin should be used locally, and potassium iodid alternating with Basham's mixture, should be administered internally.

In purulent pleural effusion (*empyema*), aspiration is of little value except for diagnostic purposes as the pus reaccumulates. Incision of the chest between the fifth and sixth ribs with the insertion of a drainage-tube is then indicated. The pleural sac should be treated then as an abscess cavity. Basham's mixture, stimulants, and quinin should be given internally in addition.

HYDROTHORAX.

Synonym.—Dropsy of the pleura.

Definition.—The effusion of fluid into the pleural cavities (bilateral), the result of a general dropsy from renal or cardiac disease. The effusion consists of a more or less clear serous fluid which occupies both pleural sacs. There are no signs of inflammation.

Symptoms.—It is accompanied by dyspnea, cyanosis, due to deficient blood aëration from compression of the lungs, and symptoms referable to the primary disease. The physical signs are those of pleural effusion.

Diagnosis.—The history, bilateral character, and the absence of pain and fever serve to distinguish it from other pleural conditions.

Prognosis.—This is controlled almost entirely by the primary cause producing the general dropsy.

Treatment.—The pleural condition will necessitate the administration of hydragogue cathartics and diuretics, and at times aspiration will be required. Dry cups over the chest may afford some relief.

PNEUMOTHORAX.

Synonyms.—Air in the pleural cavity; hydropneumothorax.

Definition.—The accumulation of air in the pleural cavities, with the consequent development of inflammation of the membranes; characterized by sharp pain, followed by rapidly developing dyspnea and cough.

Causes.—It generally results from tuberculous ulcers perforating the pleura. Abscess, gangrene, and emphysema may be causes. Perforation may take place from the pleura into the lung as the result of empyema or abscess of the chest-wall. Direct perforation from without may follow fractures of the ribs, penetrating wounds, and severe contusions.

Pathologic Anatomy.—The gas in the pleural cavity consists of oxygen, carbon anhydrid, and nitrogen in variable proportions. It may fill the pleural sac completely, compressing the lung, or may be limited by adhesions. The gas tends to excite inflammation, the resulting effusion being either serous or purulent.

Symptoms.—The onset is abrupt with sudden or sharp pain in the side, intense dyspnea, symptoms of collapse, coldness of the surface, and cold sweats. These symptoms, in many instances, follow a severe or violent paroxysm of coughing. In severe cases, the acute pain and distressing dyspnea are constant until death.

Physical Signs.—*Inspection* serves to detect enlargement of the affected side, with absent or diminished respiratory movements. The intercostal spaces are widened and sometimes bulged out so that the surface of the chest is smooth. The apex beat is displaced.

Palpation reveals diminished tactile fremitus.

Percussion yields marked changes in the resonance. Immediately after the rupture, the percussion note is hyper-resonant, or even tympanitic or amphoric in quality. If the amount of air in the pleural cavity becomes extreme, there is dullness on percussion, associated with a feeling of great resistance or density. When effusion of blood occurs, dullness is obtained over the lower part of the chest, hyper-resonant, or tympanitic percussion note over the upper portions of the chest, these sounds changing as the patient changes position.

Auscultation demonstrates several characteristic features. The normal vesicular murmur may be diminished or absent. The typical amphoric respiratory sound is heard when the fistula is open, usually associated with a metallic echo. The vocal resonance may be diminished or absent, or, rarely, it may be exaggerated, with a distinct metallic echo.

Metallic tinkling, or the bell sound, is sometimes distinctly produced by breathing, coughing, or speaking, after the development of inflammation of the pleura.

After the development of pleuritis, suddenly shaking the patient, gives rise to a splashing sensation, the succussion sound, if both air and fluid are present in the pleural cavity.

Diagnosis.—The distinctive features of this affection are the history, situation, symptoms, and physical signs, the careful consideration of which will prevent errors in diagnosis.

Prognosis.—When occurring as the result of tuberculosis, the prognosis is extremely unfavorable; rarely, the fistulous opening is closed by inflammatory action; the case then becomes one of chronic pleurisy. Cases due to other causes, are less grave but are nevertheless serious.

Treatment.—Morphin should be administered hypodermically at once, and diffusible stimulants, ammonia, alcohol, ether, etc., given at once. Aspiration of the chest followed by strapping may afford relief at times. Apart from these simple procedures the treatment is that of the primary disease.

DISEASES OF THE CIRCULATORY SYSTEM.

PHYSICAL DIAGNOSIS.

The methods employed in making a physical examination of the heart are: I. *Inspection.* II. *Palpation.* III. *Percussion.* IV. *Auscultation.*

The *Precordium* is the region overlying the heart and to which the physical examination is applied. It may be unduly prominent as the result of rickets, cardiac hypertrophy, cardiac dilatation, pericardial effusions, localized pleural effusions, empyema, and aneurysms in the region of the heart. It may be abnormally depressed as the result of spinal curvature, rickets, or the shrinking following remote pericarditis and empyema. The interspaces bulge in pericardial effusion and are retracted when adhesions form. A change in the color of the integument of the precordial region is nearly always induced by a purulent pericardial or pleural effusion on the verge of rupture.

Inspection serves to detect the exact point of the cardiac impulse, and the presence or absence of any abnormal pulsations, or any change in the form of the precordium. Normally, the impulse is visible only in the fifth interspace, midway between the left nipple and the left border of the sternum, its area covering about one inflammation, the resulting effusion being either serous or purulent.

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The *position, area,* and *force* of the impulse may be altered by disease.

The *position* may be moved to the right by left-sided pleural effusions, by chronic pulmonary or pleural disease of the right side associated with retraction, and as the result of transposition of the viscera. Displacement downward may be caused by cardiac hypertrophy, pulmonary emphysema, mediastinal growths, and aneurysm of the arch of the aorta. It may be displaced upward by a pericardial effusion or abdominal tumors. It may be moved further to the left as the result of left-sided cardiac hypertrophy or dilatation, retraction of the left side following chronic lung or pleural disease, right-sided pleural effusion, abdominal growths, and pericardial effusion.

The *area* of the impulse is enlarged by pericardial adhesions, cardiac hypertrophy, cardiac dilatation, and by thinning of the chest-walls and shrinking of the lungs from any cause. The area may be diminished by pericardial effusion and emphysema.

The *force* of the impulse may be increased by excitement, Grave's disease, certain drugs, various forms of reflex irritation, and cardiac hypertrophy. It is relatively increased by conditions that increase its area. It may be decreased by cardiac dilatation or degeneration, collapse, pericardial effusion, and emphysema.

Abnormal pulsations may be detected at times by inspection in the epigastrium, at the base of the heart, in the left axillary region, in the carotid arteries, and in the jugular vein.

Pulsation in the epigastrium may be due to aneurysm of the abdominal aorta, abdominal tumors lying over the aorta, enlargement of the right ventricle, and cardiac excitement from any cause.

Basic pulsation is usually produced by aneurysm of the arch of the aorta or cardiac hypertrophy.

Axillary pulsation (left side) may be caused by cardiac enlargement, pulsating empyema, retraction of the left side of the chest, and aneurysm.

Abnormal pulsation of the carotid arteries may be due to Grave's disease, anemia, cardiac excitement, neurotic temperament, aortic regurgitation, and disease of the vessel-walls.

Jugular pulsation may be induced by coughing, forced expiration, pericardial adhesions, and tricuspid regurgitation.

Palpation confirms the observations of inspection, and also determines the *force, frequency,* and *regularity* of the cardiac impulse.

The force of the impulse is diminished by cardiac dilatation, fatty and fibroid degenerations of the heart, emphysema, pericardial effusion, and adynamic diseases. The impulse is increased by cardiac hypertrophy, during the first stage of endocarditis and pericarditis, functional cardiac disturbances, and sthenic inflammations.

Palpation also serves to detect the shock induced by the closure of the valves. It is most marked in persons having thin chest-walls and in whom for any reason there is heightened tension either in the aorta or in the pulmonary artery.

Thrills may be also recognized by palpation and are produced by vibration of the blood in passing over a rough surface. A thrill is created only at the time the blood is passing through the orifices and is usually felt at the apex. A thrill or tremor obtained by palpation in this area is usually indicative of mitral obstruction. The apiceal thrill is presystolic in time. A thrill (systolic in time) at the second right costal cartilage is symptomatic of aortic obstruction. A systolic thrill at the second left costal cartilage points to pulmonary obstruction.

Pericardial friction may be detected by palpation. It has a to and fro movement, synchronous with the heart's action and bears no relation to respiration.

Position of the patient often alters the intensity of these abnormal phenomena. The upright posture, or slightly leaning forward serves to intensify friction, fremitus, and thrills.

Percussion will determine the boundaries of the *superficial* and *deep cardiac space,* the so-called precordium.

The *superficial cardiac space* is that portion of the heart not covered by the lung at the time of inspiration and extends from the fourth to the sixth costal cartilages and from the left border of the sternum to the apex beat. Its configuration is consequently triangular. This superficial area of dullness is increased by cardiac hypertrophy, cardiac dilatation, and pericardial effusion; it is diminished at the end of full inspiration, in emphysema, when the heart is retracted by pericardial or pleural adhesions, and when air is present in the pericardial or pleural sac.

The *deep cardiac space* (precordium) extends from the third left costosternal junction to the apex beat; from thence to the junction of the xyphoid cartilage with the sternum, the base of the triangle being formed by a line one-half inch from and parallel with the right border of the sternum. This area is increased by hypertrophy or dilatation of the heart, and pericardial effusions. It is apparently increased by shrinking of the lungs as in phthisis and in consolidation of the anterior border of the investing lung. It may be diminished in emphysema and by the presence of air in the pleural or pericardial sacs.

Auscultation indicates the character of the normal cardiac sounds and the point at which they are heard with greatest intensity, and should be thoroughly familiarized if abnormal sounds are to be fully appreciated.

The ear or stethoscope applied to the precordium distinguishes in health two sounds, separated by a momentary silence—*the short pause*, and the second sound followed by an interval of silence—*the long pause*.

The *first sound*, corresponding to the contraction of the heart — systole — is louder, longer, and of a lower pitch and a more booming quality than the second sound, and has its point of greatest intensity at the cardiac apex or a little to the left. It corresponds closely in time to the pulsations as felt in the carotid or radial arteries.

The *second sound* is shorter, weaker, and higher in pitch than the first sound, and has a clicking or valvular quality, having its point of greatest intensity at the second right costal cartilage and a little above, and corresponds to the closure of the aortic and pulmonary valves. The sound made by the closure of the tricuspid valves is best isolated at the ensiform cartilage; the sound made by the closure of the pulmonary valves, at the third left costal cartilage.

The following table, giving the phenomena and time of normal cardiac movements, will assist in recalling the physiology of the heart:

	ACTION OF HEART.	SOUND AND PAUSE.	TIME IN ONE-TENTHS OF THE HEART'S BEATS.
Systole of the heart, or ventricular systole.	Ventricles contracting, auricles dilating. Auriculo-ventricular valves (mitral and tricuspid) suddenly close and remain closed during the whole time of the first sound. Semilunar valves (aortic and pulmonary) open; movement or locomotion of heart causing the impulse or apex beat. Blood rushes out from the ventricles into aorta and pulmonary artery, and dilates these vessels and their extensions (arterial system). Blood flows slowly into the auricles from the vena cava and pulmonary veins. The pulse felt in different arteries from one-thirtieth to one-eighth of a second later than impulse.	First cardiac sound (systolic) dull and prolonged.	About four-tenths of the heart's beats, or twenty-four-sixtieths of a second.

	ACTION OF HEART.	SOUND AND PAUSE.	TIME IN ONE-TENTHS OF THE HEART'S BEATS.
Diastole of the heart, or ventricular diastole.	Ventricles dilating and receiving blood from auricles. Auricles dilating and receiving blood from veins. Auriculo-ventricular valves (mitral and tricuspid) open. Dilated pulmonary artery and aorta recoil and suddenly close the semilunar valves (aortic and pulmonic), which remain closed during the whole of the second sound and the interval of silence.	Second cardiac sound (diastole) short and sharp.	About three-tenths of the heart's beats, or eighteen-sixtieths of a second.
	Ventricles and auricles still continue to dilate — viz., receive blood. Near the close of this period the auricles, being fully dilated (filled with blood), suddenly contract and complete the dilatation of the ventricles.	Period of silence or rest.	About three-tenths of the heart's beats, or eighteen-sixtieths of a second.

The extent of surface, over which the cardiac sounds are heard, varies according to the size of the heart and the condition of the adjacent organs for transmitting sounds.

The cardiac sounds may be altered in *intensity, quality, pitch, seat,* and *rhythm,* or they may be accompanied, preceded, or followed by adventitious or new sounds, the so-called endocardial or cardiac murmurs.

The intensity is increased by cardiac hypertrophy, irritability of the heart, or consolidation of adjacent lung-structure.

The intensity is diminished by cardiac dilatation or degeneration, pericardial effusion, or emphysematous lung overlapping the heart, and during the course of adynamic fevers.

The quality and pitch of the first sound may be sharp or short and of higher pitch when the ventricular walls are thin, or have undergone fibroid change, the valves being normal; its pitch and quality are also raised during the course of low fevers. The second sound becomes duller and lower in pitch when the elasticity of the aorta is diminished or the aortic valves thickened. Either or both sounds have a more or less metallic quality in irritable heart and during gaseous distention of the stomach.

The seat of greatest intensity of the cardiac sounds is changed by displacement of the heart, pleuritic effusion, emphysematous lung overlapping the heart, pericardial effusion, and abdominal tympanites.

The rhythm is often interrupted by a sudden pause or silence, the heart missing a beat, or the sounds are irregular, confused, and tumultuous, as the result of organic changes in the cardiac muscle, valves, orifices, or vessels. A reduplication of one or both sounds of the heart may occur.

The *adventitious cardiac sounds or murmurs* are of two kinds: those produced external to the heart, as *pericardial,* exocardial, or frictional murmurs, and those made within the cardiac cavity, *endocardial murmurs.*

Pericardial murmurs, or friction sounds, are made by the rubbing upon one another of the roughened surfaces of the pericardial membrane during the early stages of inflammation. The sounds have a rubbing, creaking, or grating character, and are differentiated from a pleural friction sound by their being limited to the precordium, synchronous with every sound of the heart, and not influenced by respiration. They are distinguished from an endocardial murmur by their superficial rubbing, creaking, or grating character, and by not being transmitted beyond the limits of the heart, either along the course of the vessels, or to the left axilla or back.

Endocardial murmurs are of two kinds—to wit: *organic* and *functional.*

Functional endocardial or blood murmurs are the result of changes in the normal constituents of the blood.

Their character is soft, they are heard most distinctly at the base to the left of the sternum, during the systole, are not transmitted beyond the limits of the heart, either to the left axilla or the back, and they are associated with general anemia.

Organic endocardial murmurs are produced by blood currents pursuing either a normal or an abnormal direction.

In health there are *two direct blood currents* upon each side of the heart—to wit: the current from the left auricle to the left ventricle, the *mitral direct current;* the current from the left ventricle to the aorta, *the aortic direct current;* the current from the right auricle to the right ventricle, the *tricuspid direct current;* and the current from the right ventricle to the pulmonary artery, the *pulmonic direct current.*

When, from disease, the valves are not properly closed, the blood is allowed to flow back against the direct current, producing abnormal blood currents—to wit: when the mitral valve is incompetent, the blood flows from the left ventricle back into the left auricle during the cardiac systole, producing the *mitral regurgitant or indirect current;* when the aortic valves are incompetent, the blood is permitted to flow from the aorta into the left ventricle during the cardiac diastole, producing the *aortic regurgitant or indirect current;* when the tricuspid valves are incompetent, the blood flows from the right ventricle back into the right auricle during the systole, producing the *tricuspid regurgitant or indirect current;* when the pulmonary valves are incompetent, the blood flows from the pulmonary artery into the right ventricle, producing the *pulmonic regurgitant or indirect current.*

The *mitral direct current* occurs during the contraction of the left auricle, or just before the first sound of the heart and immediately after its second sound. The *aortic direct current* is produced by the contraction of the left ventricle, and occurs with the first sound of the heart. The *tricuspid direct current* occurs during the contraction of the right auricle, or just before the first or immediately after the second sound. The *pulmonic direct current* is produced by the contraction of the right ventricle, occurring during the first cardiac sound.

The *mitral direct* or *presystolic murmur* occurs before the first sound of the heart and immediately after the second sound. It is caused by a narrowing of the mitral orifice, has a blubbering quality, well imitated by throwing the lips into vibration by the breath, of a low pitch, and it has its seat of greatest intensity at the cardiac apex, and is not transmitted to the left axilla or to the base of the heart.

The *mitral regúrgitant* or *systolic murmur* occurs with the first sound of the heart, resulting from the failure of the mitral valves to close the mitral orifice during the systole, in consequence of which the blood flows back, or regurgitates into the left auricle. It is usually of a blowing or churning character, and has its seat of greatest intensity at the cardiac apex, being well transmitted to the left axilla and inferior angle of the left scapula.

The *aortic direct murmur* occurs with the first sound of the heart. It is caused by a narrowing of the aortic orifice, has a rough or creaking character, is of high pitch, having its seat of greatest intensity in the second intercostal space, to the right of the sternum, and is well transmitted over the cariotid artery.

The *aortic regurgitant murmur* occurs with the second sound of the heart, and is caused by the failure of the aortic valves to close the aortic orifice during the diastole, permitting the blood to flow back or regurgitate into the left ventricle. It is usually of a blowing or churning character and of low pitch, having its seat of greatest intensity over the base of the heart, and is well transmitted downward toward or below the cardiac apex. It is the only organic murmur heard in the left side of the heart which occurs with the second sound of the heart.

The *tricuspid direct murmur* occurs before the first sound of the heart and immediately after the second sound. It is caused by a narrowing of the tricuspid orifice, has a blubbering quality, and is low in pitch, having its seat of greatest intensity near the ensiform cartilage. This murmur is exceedingly rare.

The *tricuspid regurgitant murmur* occurs with the first sound of the heart, the result of the failure of the tricuspid valves to close the tricuspid orifice during the systole, thus allowing the blood to flow back or regurgitate into the right auricle. It is usually of a blowing or soft, churning character, having its seat of greatest intensity at the ensiform cartilage. This murmur is also very infrequent, and occurs mostly when the right ventricle is considerably dilated, and without the existence of any valvular tricuspid disease.

The *pulmonic direct murmur* occurs with the first sound of the heart. It is generally connected with congenital lesions. It occurs at the same instant that the aortic direct murmur occurs, and is distinguished from the latter by its not being transmitted into the carotid artery, whereas the aortic direct murmur is always thus transmitted.

The *pulmonary regurgitant murmur* occurs, like the aortic regurgitant murmur, with the second sound of the heart. This murmur is exceedingly rare, and its presence is only positively differentiated from the aortic regurgitant murmur by the absence of aortic lesions and symptoms.

SYMPTOMATOLOGY.

The Pulse.—The arterial pulsation indicates the frequency, rhythm, and force of the cardiac action and the blood-pressure. In the prenatal period the pulse varies in frequency from 120 to 140 beats per minute; in young children from 90 to 100; in healthy adults from 72 to 80; and in old age from 80 to 100. In females it is slightly greater in frequency than in males.

Tachycardia or increased frequency in the pulse may be physiologic or pathologic. It may be physiologically accelerated as the result of physical or mental exertion, fear, excitement, etc., after a heavy meal, or when the erect posture is assumed. It may be pathologically increased as the result of stimulation by drugs, fevers, heart-disease, reflex irritation, exophthalmic goiter, and various morbid conditions at the base of the brain interfering with the function of the pneumogastrics. It may occasionally arise as an independent affection, no cause being demonstrable.

Bradycardia or infrequency of the pulse, slow pulse, may be observed in jaundice, atheroma, lesions of the cerebral centers, especially such as irritate the pneumogastric nerves at their origin, fatty degeneration of the heart, aortic stenosis, in the terminal stages of certain febrile affections, and after the ingestion of drugs such as digitalis, aconite, and opium. Occasionally this condition may be observed in health without obvious cause and as a purely physiologic phenomenon.

The rhythm of the pulse is also subject to variations (*arhythmia*).

The *intermittent pulse* may be observed as the result of excessive eating, the habitual use of tobacco, coffee, and tea, exercise, mental excitement, myocardial disease, and reflex irritation such as produced by constipation, dyspepsia, lithemia, hypochondriasis, etc.

The *irregular pulse* may be due to the same causes as the preceding. As a pathologic condition, it is most often encountered in organic cardiac disease, especially that which gives rise to mitral regurgitation.

The *dicrotic pulse* is one in which the first impulse is quickly followed by another impulse or secondary wave. It owes its production largely to conditions which relax the arterial walls and lower the tension, especially adynamic affections such as typhoid fever.

Pulsus paradoxus is that condition of the pulse in which the pulse-wave becomes small and feeble during inspiration; it may occur in health but is rather common as the result of pericardial adhesions.

Water-hammer or *Corrigan's pulse* is that pulse which is charterized by a short, sharp, strong impulse which seems to collapse under the examiner's fingers. It is best detected by holding the arm up, and is diagnostic of aortic regurgitation during compensation.

A *full pulse* is one in which the volume is large and is encountered in the robust and plethoric; a *small pulse* has a weak beat and small volume, and is observed in exhausting or debilitating conditions, aortic stenosis, mitral stenosis, myocarditis, Bright's disease, acute peritonitis, and during a chill.

A *strong pulse* has a strong impulse and very little compressibility, and is found in robust individuals and in cardiac hypertrophy; a *weak pulse* is the direct opposite and attends asthenic affections.

Tension of the pulse expresses the degree of distention of the arteries, or blood-pressure. The pulse may be hard or soft. A hard pulse is one of high tension or in which the contractile power of the arterial walls is great. The artery consequently remains continuously full between the beats. Among its causes may be mentioned plethora, increased cardiac action with contraction of the arterioles, capillary obstruction from various causes, cardiac hypertrophy, arteriosclerosis, interstitial nephritis, gout, lithemia, uremia, lead-poisoning, pregnancy, anemia, apoplexy, brain tumor, etc. A *soft pulse* is one of low tension and easily compressible, due directly to a lowered tone of the vessel-walls. This may occur physiologically. As an abnormal condition it is encountered in asthenic affections such as typhoid fever, cardiac degeneration, collapse, etc. It may occur in obese individuals and after diarrhea, warm baths, hot applications, hot drinks, and copious urination.

Palpitation of the heart consists of abnormal rapidity with fluttering and tremor of the organ, of which the patient is conscious. It is usually purely functional in origin and may be traced in most cases to gastrointestinal disturbances, excitement, hysteria, overwork, etc. It may be also due to organic heart-disease, exophthalmic goiter, and anemia.

Dropsy.—Serous infiltration of the cellular tissues and cavities of the body frequently is indicative of heart disease, especially when it is bilateral. When localized to certain regions as the ankles it is termed *edema,* and when generalized it is known as *anasarca.*

Cyanosis is the term applied to blueness of the body-surface and is due to deficient oxidation of the blood as the result of local or general circulatory disturbances. It therefore accompanies various forms of chronic heart disease. It may be congenital as the result of cardiac malformations.

Pain in the precordial region may be due to disease of the heart or pericardium, neuralgia, pleurodynia, myalgia, localized pleurisy, periositis, or abscess. Cardiac disease may induce acute, excruciating pain in the epigastrium. Disturbances of rhythm, valvular disease, and angina pectoris are common causes of cardiac pain. Pericarditis is attended by paroxysmal pain over the heart which may radiate to the left shoulder and down the arm, being increased by pressure, movement, and respiration. Inflammation, atheroma, or aneurysm of the aorta may be the cause of pain in the precordial region.

Dyspnea is also a symptom of heart-disease and may be due to exertion, or it may occur paroxysmally. Orthopnea is observed in grave cases, as is also rhythmic dyspnea or Cheyne-Stokes breathing.

Cerebral symptoms may be induced by cardiac disease. Among these may be mentioned vertigo, faintness, dullness, languor, stupor, moderate delirium, coma, chorea, epileptiform convulsions, etc.

Gastrointestinal symptoms such as dyspepsia, flatulence, nausea, vomiting, and similar manifestations of gastric congestion may attend organic heart disease.

ACUTE PERICARDITIS.

Definition.—An acute fibrinous inflammation of the pericardium; characterized by slight fever, pain, precordial distress, and disturbed cardiac action and circulation. If the inflammation be limited to the parietal or visceral layer, or to a part of either, it is termed *partial* or *circumscribed* pericarditis; if it involve the whole of both surfaces, it is termed *general* or *diffused* pericarditis. The inflammation may be primary or secondary.

Causes.—Primary pericarditis resulting directly from cold and exposure or injuries is rare. Secondary pericarditis follows, or is associated with, rheumatism, influenza, scarlatina, variola, puerperal fever, tuberculosis, septicemia, Bright's disease, gout, scurvy, diabetes, and with pneumonia and pleuro-pneumonia, particularly in alcoholics. Microörganismal infection is the direct cause.

Pathologic Anatomy.—The structural changes in this affection are similar to inflammation in other serous membranes. These changes present themselves as acute plastic or dry pericarditis or pericarditis with a sero-fibrinous, hemorrhagic, or purulent effusion. The earliest change is that of hyperemia, most marked on the visceral layer, giving it a dull red appearance, which is followed by the exudation of lymph in scattered and irregular patches causing the membrane to appear rough and shaggy (*dry pericarditis*). Later, there is an effusion poured out which may be serofibrinous, hemorrhagic, or purulent. In sero-fibrinous pericarditis the effused material consists largely of straw-colored fluid which varies in quantity from a few ounces to one or two pints or more. The extravasation of blood into the sac from any cause during the affection gives rise to the hemorrhagic form, and the purulent variety results from pyogenic infection of the membrane. When the serum is deficient in the exudate and fibrin predominates, the effusion is extremely scant and the term fibrinous pericarditis is applied. Its onset is less acute and the tendency to form adhesions is very great. Varying grades of myocardial inflammation are encountered in combination with pericarditis.

Symptoms.—Acute pericarditis may be well-marked and still present none of the characteristic subjective symptoms. It usually begins with rigors, fever of the remittent type, frequently nausea and vomiting, precordial distress and tenderness, acute shooting pains, increased by breathing and coughing; dry, suppressed cough; increased cardiac action, and sometimes violent palpitation. An attack of pericarditis secondary to an existing disease presents no marked symptoms other than those mentioned to indicate its onset. Attacks of nausea and vomiting occuring during the course of rheumatism, pneumonia, pleurisy, and nephritis should always call attention to the heart. The duration of this early stage is from a few hours to a day or two.

During the stage of effusion, the symptoms vary with the amount of effusion and the rapidity with which it is formed. There are precordial oppression, tendency to syncope, dyspnea at times amounting to orthopnea, dysphagia, hiccough, nausea, vomiting, feeble irregular pulse, and sometimes melancholia, delirium, or acute maniacal excitement.

Absorption is usually rapid, but the heart remains irritable for quite a long period. If absorption does not occur and the fluid accumulates continuously without destroying life at the time, the pericardial sac becomes dilated, and chronic pericarditis is produced.

A purulent effusion in the pericardial sac is evidenced by irregular fever, chills, sweats, and leukocytosis in addition to the symptoms already given.

Physical Signs.—*Inspection* during the early stage shows excited cardiac action as evidenced by the impulse. During the effusion stage, the impulse is feeble, undulatory, or absent,; it is usually displaced upward, very rarely downward, the precordium bulges, and the abdomen protrudes when the effusion is large.

Palpation during the early stage serves to detect an excited or tumultuous impulse, and in very rare instances pericardial friction fremitus. During the effusion stage, the impulse is feeble or absent and when present is considerably displaced. Tenderness may be elicited.

Percussion is normal in the beginning of the disease but as the effusion forms the cardiac dullness becomes enlarged vertically and laterally. If the accumualtion of fluid is considerable, the dullness assumes a triangular shape with the base on a line with the sixth or seventh rib extending from the right of the sternum to the left of the left nipple and the apex at the sternal attachment of the second rib or higher. The shape of the dullness is sometimes altered by changing the position of the patient.

Auscultation at the onset, reveals excited cardiac action and usually an exocardial murmur or friction sound, synchronously with the cardiac sounds and uninfluenced by respiration but often increased by pressure with the stethoscope. Later as the effusion forms, the cardiac sounds are feeble and deep-seated at the apex, becoming louder and distinct toward the cardiac base. The friction sound is sometimes heard at the base. As absorption progresses, the friction sound returns, being replaced shortly by the normal heart-sounds.

Diagnosis.—*Acute endocarditis* may be distinguished from acute pericarditis by the absence of friction sound and triangular dullness and by the presence of soft systolic or diastolic murmurs heard best over one of the valve-points.

Cardiac hypertrophy is unattended by acute symptoms, friction sound, or evidences of effusion. The onset is less sudden. The impulse is strong and the sounds are loud.

Cardiac dilatation is characterized by enlargement of the area of dullness downward, undulatory impulse, and clear and distinct heart sounds. There is no friction sound.

Hydropericardium is attended by the physical signs common to pericardial effusion, but at no time is a friction sound obtained on examination. The history of other dropsies and their underlying diseases aids in making the diagnosis.

Prognosis.—This is controlled by the severity of the inflammation, its causes, and the coexisting conditions. Pericarditis with slight effusion is frequently overlooked and often terminates, favorably without being detected. Simple sero-fibrinous pericarditis without complications and under proper treatment ends in recovery in from one to three weeks. In debilitated subjects the disease is prolonged. The rapid effusion of large quantities of fluid may cause sudden death. Purulent effusions are usually fatal. Fibrinous pericarditis is attended by adhesions and subsequent changes in the heart-muscle. Relapses are not infrequent.

Treatment.—Absolute rest in bed with mental quiet is necessary. Death has followed neglect of this simple precaution. Milk diet should be prescribed. Local applications are especially valuable in the early stage: In vigorous patients leeches or wet cups applied to the precordium followed by ice-poultices or iced-compresses; in the feeble and debilitated, dry cups to the precordium, followed by poultices. Blisters are also very beneficial. Cold applications by means of Leiter's coil or ice-bags are sometimes more comfortable than the foregoing procedures. Occasionally heat is more serviceable. The hypodermic injection of morphin sulphate, gr. ¼ (0.0165 Gm.), and atropin sulphate, gr. 1-150 (0.00044 Gm.), serves to relieve pain and quiet the heart.

Mercury is often of value in relieving the gastrointestinal symptoms and in lessening the pericardial inflammation. The following formula is employed with benefit:

℞.	Hydrargyri chloridi mitis gr. ⅓	.022 Gm.
	Sodii bicarbonat. ....gr. ij	.13 Gm.
	Sacchar. lactis ....gr. ij	.13 Gm.

M. S.—To be taken dry on the tongue every two hours until free action of the bowels is obtained.

The late Dr. Pepper recommended the following combination:

℞.	Pulv. digitalis .... Mass. hydrargyri. ....āā gr. x	āā .6 Gm.
	Pulv. opii ....gr. v	.3 Gm.
	Quininæ sulph. ....gr. xxx	2. Gm.

M.—Ft. mass et div. in pil. No. xx.
S.—One pill three or four times daily.

In young, vigorous patients, the excited cardiac action may be controlled early in the disease by small doses of aconite or veratrum viride; in adults, the aged, and feeble individuals, digitalis, in doses sufficient to steady the heart, but not to stimulate too forcibly, should be employed. Quinin, strychnin, alcohol, and ammonia are of value in all cases. In secondary cases, except those due to rheumatism, cardiac sedatives should be avoided; the treatment recommended for the primary condition should be continued and combined with the measures indicated for the pericarditis.

During the stage of effusion, the diet should still be liquid, and stimulants should be continued to maintain the heart's action. Ammonium carbonate, solution of ammonium acetate, potassium acetate, potassium carbonate, quinin sulphate, and saline purgatives are also indicated. Diuretics may be employed but *diaphoretics such as pilocarpin are contraindicated.* If the effusion gives rise to marked pressure symptoms, tapping should be performed either in the fossa between the ensiform and costal cartilages on the left side, or in the fifth left interspace near the junction of the sixth rib with its cartilage. Blisters and potassium iodid aid in absorption of the exudate.

When the exudate is purulent, incision of the chest wall and drainage of the pericardial sac is indicated. The toxemia is profound and tonics and stimulants should be given very freely.

CHRONIC PERICARDITIS.

Synonym.—Adhesive pericarditis.

Definition.—A chronic inflammation of the pericardium, with either distention of the sac by fluid, or adhesions of the pericardium (adherent pericardium); characterized by impaired cardiac action and disturbances of the circulation.

Cause.—The affection is always secondary to an acute attack.

Pathologic Anatomy.—If the fluid is absorbed, the pericardial surfaces become agglutinated by several layers of lymph, which serve to increase the thickness of the sac-wall half an inch or more. Often the outer surface of the pericardium becomes adherent to the chest-walls. If the fluid is not absorbed, it may continue to accumulate, distending the sac in all directions, displacing the diaphragm, and interfering with the functions of adjacent viscera; or a low grade of septic inflammation may supervene with the formation of a purulent effusion (empyema of the pericardium), the disease terminating fatally after a varying period.

Symptoms.—Precordial pain and distress are prominent symptoms. Cardiac action is irregular and feeble, and dyspnea, worse on movement, with other signs of embarrassed circulation is present. When agglutination of the walls occurs, there arises a great tendency to pulmonary inflammation.

Physical Signs.—*Inspection* detects bulging of the precordium and displacement of the impulse if the effusion is yet present. If the pericardium is adherent to the chest-wall, depression of the precordium and recession of the intercostal spaces (*systolic dimpling*) and epigastrium with every systole, will occur. The interspaces are narrowed and the impulse is more diffuse but displaced and uninfluenced by deep inspiration.

Palpation serves to confirm inspection. In the presence of an effusion, the impulse is displaced and feeble or absent; if adhesions exist, the impulse is displaced and tumultuous. A friction fremitus may occasionally be obtained.

Percussion will be of service in outlining dullness corresponding to the effusion if any is present. If adhesions only exist, the cardiac dullness is but slightly modified.

Auscultation reveals feeble and deep-seated cardiac sounds at the apex, and loud and more distinct sounds at the base if there is any effusion. If adhesions are present the cardiac sounds are unaltered and a rough friction sound or exocardial murmur may be obtained.

Treatment.—The cardiac action should be carefully watched and maintained by stimulants, as advised in the acute form. Blisters, potassium iodid, purgation, and other means calculated to absorb inflammatory exudates should be employed. Paracentesis may be necessary. Incision is indicated if the effusion becomes purulent.

HYDROPERICARDIUM.

Synonym.—Pericardial dropsy.

Definition.—The accumulation of water in the pericardial sac, without inflammation; characterized by precordial distress, disturbed cardiac action, dyspnea, and dysphagia.

Causes.—It is always a secondary affection being due to heart disease, Bright's disease, pneumothorax, pressure of an aneurysm or other mediastinal tumor, or disease of the cardiac veins.

Pathology.—A pericardial effusion is formed without any evidences of inflammatory changes. The fluid may range in quantity from an ounce to one or two pints and is usually clear, yellowish, and straw-colored and of an alkaline reaction.

Symptoms.—Manifestations of dropsy in other parts of the body, or anasarca, are present. The pericardial involvement is indicated by disturbances of the heart's action, dyspnea, dysphagia, dry cough, and other manifestations of cardiac embarrassment.

Physical Signs are those of pericardial effusion from other causes; the friction sound, however, is never obtained.

Diagnosis.—A differentiation between this affection and pericarditis with effusion can be made only by consideration of the history and the results of aspiration.

Prognosis.—The outlook depends entirely upon the nature of the underlying disease.

Treatment.—The effusion calls for paracentesis if the cardiac action is seriously disturbed. The major portion of the treatment should be directed towards the original cause of the dropsy.

ACUTE ENDOCARDITIS.

Synonyms.—Valvulitis; exudative endocarditis.

Definition.—An acute fibrinous inflammation of the serous membrane lining the cavity of the heart and particularly its valves, in severe cases the chordæ tendinæ being involved, resulting in changes in the valves or orifices of the heart, or both; characterized by cough, dyspnea, disturbed cardiac action, nausea, vomiting, and more or less marked febrile reaction. Acute endocarditis occurs in two distinct forms: *plastic* or *simple exudative* endocarditis; *ulcerous* or *diphtheritic* endocarditis. The ulcerative form is considered under a separate heading. (See Malignant Endocarditis.)

Causes.—Acute simple endocarditis is usually secondary to some other affection, particularly acute articular rheumatism (especially in young people), chorea, pleurisy, pneumonia, pericarditis, Bright's disease, and the infectious fevers such as scarlatina, influenza, and diphtheria. Gonorrhea is an occasional cause. Cachectic states such as accompany tuberculosis and cancer are predisposing causes. It may be secondary to chronic endocarditis.

Pathology.—Acute simple endocarditis may occur prenatal as well as postnatal. In the former class of cases, the right side of the heart is usually involved, while in those instances observed after birth, the disease is most often limited to the left side. While the disease may attack the entire lining membrane of the heart it is especially marked at the valvular portions of the endocardium. The earliest change is that of hyperemia of the membrane rendering it red and swollen. As the inflammatory exudate is thrown out the surface of the valves become roughened and warty excrescences are formed. These verrucose formations are to be found on the auricular surface of the mitral valve and on the ventricular surface of the aortic valve at the line of contact of their leaflets, usually from 1 to 2 mm. from their free margin. These vegetations are produced by a proliferation of the cells of the adventitia and of the external connective-tissue; fibrin from the blood is deposited on the formations, thus serving to increase their size. The excrescences are friable and may be easily detached or broken off and carried in the blood stream as emboli, to various parts of the body, particularly the left side of the brain, the kidneys, and the spleen. If retained in position, fibrous tissue is eventually formed; the valves become thickened and contracted producing chronic endocarditis. The leaflets may then become the seats of various infiltrations.

Symptoms.—Occurring as it does, in the course of some other disease the subjective symptoms of acute simple endocarditis are usually masked by the manifestations of the primary condition, until disturbances of the circulation direct attention to the heart. Increase of temperature, precordial distress, cough, slight dyspnea, and more or less persistent vomiting may be present. The action of the heart is increased and often tumultuous. The carotids throb and there are noises in the ear. As the inflammation progresses, the cardiac action and pulse become less frequent, and venous stasis and more or less pulmonary congestion occur. The attack lasts from one to three weeks.

Physical Signs.—*Auscultation* reveals a change in the character of the sounds (prolongation) and sometimes the development of a murmur corresponding to the affected valve.

Diagnosis.—In all diseases in which endocarditis is liable to occur, physical examination of the chest should be made at frequent intervals as the symptoms are by no means distinctive and the diagnosis is made largely by the physical signs.

Pericarditis is distinguished from endocarditis by the character of the physical signs. In pericarditis, the murmur or friction sound is heard with either cardiac sound, is near to the ear, and is influenced by pressure of the stethoscope, besides being associated with more or less alteration in the size and shape of the cardiac dullness, and is not transmitted, while in endocarditis the murmur takes the place of, or is associated with the cardiac sounds, and is transmitted to points beyond the precordium, with absence or change in size and form of the cardiac dullness on percussion.

Embolism in the course of endocarditis produces an additional group of symptoms, the presence of which may give rise to confusion. Embolism of the kidneys causes sudden, deep-seated lumbar pain, with albuminuria and even hematuria; embolism of the brain, sudden palsies and sudden disturbance of consciousness; of the spleen, sharp pain and tenderness in the splenic region; of the skin, petechial or purpuric spots.

Prognosis.—Acute simple endocarditis without complications is not dangerous to life but the affected valve usually remains damaged and later becomes the seat of chronic endocarditis.

Treatment.—Absolute rest in bed and liquid diet are essential. Leeches, wet cups, ice, or poultices applied to the precordium may be of value. If the heart is weak and irregular digitalis in moderate doses may be employed. Purgation by salines should be obtained early in the disease. The free administration of alkalies such as ammonium carbonate, potassium carbonate, and potassium acetate until the urine is alkaline, may serve to prevent permanent changes in the valves. If the alkalies fail and the inflammation shows a tendency to linger, mercury should be administered. Dyspnea may usually be relieved by the administration of opium or morphin. If symptoms of embarrassment of the circulation appear such as marked dyspnea, cyanosis, and edema, strychnin, atropin, nitroglycerin, digitalis, ammonium carbonate, and similar heart tonics are indicated. After acute symptoms have subsided absorption of the exudate may be brought about to some extent by the free use of potassium iodid.

MALIGNANT ENDOCARDITIS.

Synonyms.—Ulcerative endocarditis; septic, mycotic, and diphtheritic endocarditis.

Definition.—An acute septic inflammation of the lining membrane of the heart, with a strong tendency to ulceration; characterized by depression of the vital forces with more or less cardiac distress.

Causes.—Microörganismal infection is the primary cause but a specific organism has as yet not been isolated. It may follow pneumonia, erysipelas, septicemia, puerperal fever, influenza, meningitis, gonorrhea, or acute rheumatism.

Pathologic Anatomy.—The changes are those of acute endocarditis up to the development of the thickening of the endocardium lining the valves, and the development of the vegetations. Instead of the poison spending its force and the chronic condition obtaining, a process of softening, ulceration, development of abscesses, and perforation of leaflets follows, resulting in loss of structure, general septic infection, and the development of emboli, which lead to infarctions, in the brain, kidney, spleen, eye, or skin.

Symptoms.—The septic intoxication is manifested by headache, restlessness, delirium of varying degrees, dry coated tongue, sordes on the lips and on the gums, nausea, vomiting, constipation or diarrhea, leukocytosis, irregular fever, rigors, and sweats. The heart's action is rapid, irregular, and weak, and the pulse is compressible. The spleen is enlarged and albuminuria is present. Paroxysmal dyspnea and cyanosis are common symptoms. The patient frequently experiences a sense of impending danger, great anxiety, and terror. The occurrence of embolism is marked by additional symptoms referable to the organ affected. If the brain, rapidly developing palsies with disorder of consciousness; if the kidneys, deep-seated lumbar pains with hematuria or disordered urinary flow; if the spleen, pain and tenderness of the splenic region with increase of temperature record.

Physical Signs.—*Auscultation* reveals the replacement of the normal, booming, muscular, first sound by a feeble, irregular cardiac pulsation. Generally a murmur may be detected but it is subject to great variations, and may be absent.

Diagnosis.—This is extremely difficult; the occurrence of septic phenomena together with symptoms of cardiac embarrassment in the course of various affections mentioned under causes, is highly suggestive.

Typhoid fever is less acute; the fever is more regular; the abdominal symptoms are more marked; a roseolar eruption is present, the leukocytes are not increased; the Widal reaction is obtained; and typhoid bacilli are found in the stools.

Prognosis.—The termination is almost invariably fatal in from one to eight weeks or more.

Treatment.—This is very unsatisfactory. Nutritious food and stimulants such as quinin, iron, alcohol, strychnin, digitalis, and nitroglycerin should be freely used to support the patient and to maintain the heart's action. Sponging will serve to reduce the temperature and render the patient more comfortable. Belladonna plaster over the precordium is beneficial but other applications seem only to increase the distress. Antistreptococcus serum (20 Cc. injected daily) has been employed by some observers with success and considering the prognosis of this affection, it is worthy of a fair trial.

CHRONIC ENDOCARDITIS.

Synonyms.—Sclerotic endocarditis; interstitial endocarditis; chronic valvular disease.

Definition.—Alterations in the cardiac valves or orifices, rendering the former incapable of properly closing the orifices (regurgitation), or causing the narrowed orifice to interrupt the blood current in its normal movement (stenosis).

Varieties.—I. Mitral regurgitation. II. Aortic regurgitation. III. Tricuspid regurgitation. IV. Pulmonary regurgitation. V. Mitral obstruction. VI. Aortic obstruction. VII. Tricuspid obstruction. VIII. Pulmonic obstruction.

Causes.—The great majority of cases are the result of an attack of acute endocarditis following rheumatism, chorea, or the infectious diseases. A chronic endocarditis from the onset may be caused by alcoholism, syphilis, gout, or excessive muscular labor. Chronic Bright's disease is also an exciting cause. In elderly people, chronic endocarditis may often be due to atheromatous or fibroid changes.

Compensation.—The alterations in the systemic blood-supply caused by the valvular defects of chronic endocardial inflammation are such that, if continued, the integrity of the body is threatened. To overcome the impaired functions of the valves and to maintain the general circulation, the heart increases in size and strength (*compensatory hypertrophy*). The period in which this occurs is called the *period of compensation;* its duration is indefinite. It may be recognized by the physical signs of valvular disease without any symptoms of disturbed circulation. Anything which disturbs the equilibrium as it now exists, such as acute diseases and excessive work, leads to *ruptured compensation,* a condition attended by cyanosis, dyspnea, edema, gastric, hepatic, and renal disturbances, and often death. The object in the treatment of all forms of chronic valvulitis is to obtain compensation and to prevent its failure or rupture.

MITRAL REGURGITATION.

This form of valvular disease is also termed mitral insufficiency, and is the most frequent variety of valvular heart-disease.

Pathologic Anatomy.—The most common conditions observed are more or less contraction and narrowing of the tongues of the valves, with irregular thickening and rigidity; atheroma or calcification of the segments; laceration of one or more segments; adhesion of one of more segments to the inner surface of the ventricle; thickened and stiffened, or ruptured, *chordæ tendineæ*, and also contraction and hardening of the *musculi papillares*.

As a result of the regurgitation or leakage of the blood back into the left auricle, there is a dilatation of the auricle, followed by slight cardiac hypertrophy. Ventricular hypertrophy occurs after a time from the increased number of the cardiac contractions. If, as is eventually the case, the left auricle is unable to overcome the backward flow of blood, it dilates and the lungs become congested. The right ventricle is then forced to perform more work and hypertrophies. Hypertrophy of the right ventricle is followed by that of the left ventricle. In the event of its failure to overcome the backward flow, it (right ventricle) also dilates and the tricuspid valve becomes insufficient.

Symptoms.—Insufficiency of the mitral valves soon leads to cardiac hypertrophy, in order to compensate for the diminished amount of blood sent onward by the ventricular systole. This condition causes quickened and strong pulse with some shortness of breath on severe exertion. When compensation ruptures, it is manifested by precordial distress, cough, dyspnea, feeble, soft, rapid, irregular pulse, pulmonary congestion, edema of the limbs, ascites, general cyanosis, hepatic congestion, and scanty and albuminous urine; all of which symptoms may present themselves in varying degrees. When extreme and compensation is not again brought about, death is the result.

Physical Signs.—*Inspection* shows displacement of the apex beat downward and to the left. In children and youths, bulging of the precordium and increased cardiac impulse are present. In emaciated individuals, an auricular impulse may be observed to the left of the pulmonic area in the second interspace.

Palpation serves to confirm inspection. The displaced cardiac impulse is forcible and diffused in the early stage; as compensation fails, the impulse becomes feeble or absent.

Percussion shows an increase in the area of cardiac dullness transversely and vertically.

Auscultation reveals a systolic or blowing murmur, audible in the mitral area and transmitted to the apex, left axilla, and under the angle of the scapula. It may occur with or take the place of the first sound of the heart, the second sound being markedly accentuated, particularly in the pulmonic area.

Prognosis.—So long as the compensating hypertrophy can be maintained, the prognosis is not unfavorable; when dilatation supervenes, however, the patient soon perishes, either from congestion of the lungs or dropsy and exhaustion.

AORTIC REGURGITATION.

This is also termed aortic insufficiency, and occurs next in frequency to mitral insufficiency.

Pathologic Anatomy.—The valves or segments adhere to the walls of the aorta, or a segment is lacerated or perforated, or, more commonly, the segments are shrunken, deformed, and rigid, permitting regurgitation of the blood. These deficiencies are usually associated with more or less dilatation of the orifices.

The inability of the aortic valves to completely close the aortic orifice at the proper moment allows the blood that should go onward to flow back into the left ventricle, and the normal flow of blood from the left auricle continuing, causes overfilling of the ventricle, which results in a dilatation of its cavity, and the extra effort of the ventricle to empty itself results in hypertrophy of the walls. In no other condition does the dilatation and hypertrophy of the cardiac walls reach such a degree. The older writers named this enormous enlargement of the heart "*cor bovinum*."

Symptoms.—As long as the cardiac hypertrophy is just sufficient to compensate for the valvular condition, there are no symptoms, but as the muscle-walls continue to increase symptoms of cardiac hypertrophy present themselves, such as forcible cardiac action, with marked pulsation of all the vessels including the capillaries, the characteristic forcible and receding pulse ("water-hammer pulse," or "Corrigan pulse"), headache, insomnia, tinnitus aurium, congestion of the eyes and face, etc. Precordial pain is usually present in aortic disease. It may be a sensation of constriction in the cardiac region or it may consist of sharp, shooting pains extending to the arms—anginoid attacks. As soon as the slightest failure of compensation occurs, the cardiac action becomes excessive and distressing. Palpitation is present and causes anxiety and fear on the part of the patient. When there is complete rupture of compensation, there develop, either gradually or rapidly, dyspnea, increased on exertion, cough, cyanosis, hepatic enlargement, renal congestion with scanty, albuminous urine, ascites, and dropsy. If mitral insufficiency is now superadded, general venous stasis and death rapidly follow. Sudden death is most frequent in this form of valvular heart-disease.

Physical Signs.—*Inspection* shows that the cardiac impulse is forcible and displaced downward and to the left. The pulsation is visible far beyond the normal apex.

Palpation confirms inspection. It may at times serve to detect a diastolic thrill over the base of the heart and the adjacent large vessels. The Corrigan pulse and the capillary pulse are recognized by palpation.

Percussion serves to demonstrate an increase in the area of cardiac dullness downward and to the left. Occasionally it is increased upward and to the left of the sternum as the result of hypertrophy of the left auricle.

Auscultation reveals characteristic alterations in the heart sounds. The first sound is forcible; the second sound is replaced or associated with a churning, rushing, or blowing murmur of low pitch, well heard at the second right costal cartilage (aortic area) but most distinct at the junction of the sternum and the fourth left costal cartilage. It is transmitted downward and toward the apex. A presystolic rumbling murmur (Flint murmur) may occasionally be heard over a limited area at the apex.

Prognosis.—Sudden death is more liable to occur in this than in any other form of chronic valvular disease. As long, however, as the compensating hypertrophy is intact, it is compatible with quite an active life. The outlook is largely influenced by the condition of the arterial walls. Obstruction from any cause induces rupture of compensation. Next to tricuspid regurgitation it is the most serious of all chronic valvular conditions. Overexertion influences it unfavorably.

TRICUSPID REGURGITATION.

Pathologic Anatomy.—This form of valvular insufficiency is either associated with right-sided cardiac dilatation from pulmonary obstruction, or is the result of mitral disease. The tricuspid orifice is dilated in the majority of cases; occasionally the segments of the valves are contracted or adherent to the ventricle. It may be due to prenatal endocarditis or to endocarditis in childhood.

Symptoms.—The manifestations of this condition are all the result of venous stasis, and include jugular pulsation synchronous with the heart's action, hepatic, renal, and pulmonary congestion, pulsation of the liver, cyanosis, dyspnea, and obstinate dropsy. These symptoms are superadded to those of the primary or associated conditions.

Physical Signs.—*Inspection* detects a diffused, wavy, cardiac impulse, synchronous with the heart's action and uninfluenced by respiration, more or less prominent hepatic pulsation, cyanosis, dyspnea and edema. Venous pulses in Jugulars

Palpation shows the cardiac impulse to be feeble and extended downward.

Percussion reveals hypertrophy of the right ventricle as is evidenced by the increased area of cardiac dullness to the right of and below the sternum.

Auscultation serves to elicit a blowing systolic murmur most intense at the junction of the fourth and fifth ribs with the sternum. It is distinct over the xyphoid appendix becoming feeble or lost in the left axillary region. It is often associated with a mitral systolic murmur. The pulmonary second sound is weak.

Prognosis.—This is the most unfavorable variety of chronic valvular disease of the heart. Dropsy, dyspnea, and cyanosis persist in spite of treatment. The failure to restore compensation results in death.

PULMONIC REGURGITATION.

Pathologic Anatomy.—Insufficiency of the pulmonary valves is of rare occurrence, but when present, the changes correspond, more or less, with those described under aortic regurgitation.

Symptoms.—Most of the symptoms are referable to dilatation of the right side of the heart and consequent pulmonary congestion, such as dyspnea, cyanosis, distention of the superficial vessels, palpitation of the heart, precordial distress, suffocative attacks, and dropsy.

Physical Signs.—*Percussion* shows extension of the cardiac dullness to the right of the sternum.

Auscultation reveals a loud blowing diastolic murmur most distinct at the junction of the third left costal cartilage and the sternum.

Prognosis.—Death results, sooner or later, from dropsy and exhaustion.

MITRAL OBSTRUCTION.

Mitral obstruction or stenosis is not so frequent as regurgitation, and is very often associated with the latter. It may be encountered as a single affection in young persons, especially females. It may be due to acute or chronic endocarditis, or it may occasionally be congenital.

Pathologic Anatomy.—Mitral stenosis is caused by deposits around the orifice, or else the segments of the valves are "glued together by their margins," leaving but a funnel-shaped opening, the so-called "buttonhole" mitral valve. Vegetations on the valves lead to more or less obstruction to the blood-current.

Symptoms.—The obstructed mitral orifice gives rise to hypertrophy of the left auricle which in time is followed by dilatation. The symptoms are usually unobservable until compensation ruptures, which is manifested by small, irregular, and feeble pulse, dyspnea, cough, bronchorrhea, and dilatation of the right side of the heart soon leading to general venous stasis, dropsy, and death.

Physical Signs.—*Inspection* shows nothing abnormal until hypertrophy of the left auricle occurs when an undulatory impulse is observed over its area. Bulging over the lower part of the sternum may be present.

Palpation serves to recognize a presystolic thrill near the apex (in the fourth or fifth interspace within the nipple-line). When cardiac dilatation occurs, a diffused, feeble, and irregular cardiac impulse is felt near the xyphoid appendix.

Percussion may demonstrate increased area of cardiac dullness on the right side.

Auscultation elicits nothing abnormal in the first or the second sound except possibly disturbances of rhythm. A blowing, sometimes rasping, sound is heard, immediately after the second sound, and immediately before the first sound begins, which is especially characteristic. This presystolic murmur is heard most distinctly in the mitral area lessening in intensity toward the base. It is not transmitted but is occasionally heard in atypical regions such as the axilla and angle of scapula. The second sound in the pulmonary area is accentuated. As the condition reaches its terminal stage, the murmur may disappear and the first sound becomes snappy in character. With the onset of dilatation all the heart-sounds become enfeebled.

Prognosis.—The outlook depends upon whether auricular hypertrophy occurs and how long it is maintained. Under favorable circumstances, mitral stenosis is compatible with a long and rather active life.

AORTIC OBSTRUCTION.

Pathologic Anatomy.—Stenosis of the aortic orifice is caused by the projection of the valves inward, and their becoming rigid and thickened, atheromatous or calcareous, so that they cannot be pressed back by the blood, but remain constantly in the current of the circulation. Occasionally the valves are covered with fibrinous masses, the opening into the artery being thus more or less completely closed, or the segments may be adherent by their lateral surfaces, leaving a central opening, which may be so contracted as to permit the passage of only the smallest probe. Aortic stenosis is nearly always a disease of advanced life, and is associated with the arterial changes of age. Uncomplicated cases are rare. Aortic disease is not nearly so often of rheumatic origin as mitral disease.

Symptoms.—Hypertrophy of the left ventricle rapidly supervenes upon aortic stenosis, and so long as the cardiac hypertrophy is just sufficient for compensation, there will be no subjective symptoms, many cases of stenosis being discovered only when the individual is examined for insurance or other purposes. The pulse is small, slow, and hard. When, however, the compensatory hypertrophy begins to fail, the supply of blood to the brain is insufficient in many cases, and pallor, with attacks of vertigo, syncope, or slight epileptiform seizures occur; finally, as dilatation of the left ventricle and incompetence of the mitral valve result, there occur pulmonary congestion, dyspnea, and general venous stasis, the pulse being soft and feeble.

Physical Signs.—*Inspection* serves to detect displacement of the apex beat downward and to the left varying with the degree of hypertrophy.

Palpation confirms inspection. The impulse is strong in the early stage, becoming feeble with the onset of dilatation.

Percussion shows a slight increase in the cardiac dullness, vertically, the transverse dullness being but slightly increased.

Auscultation reveals characteristic changes in the heart-sounds. The first sound of the heart is replaced or associated with a harsh, rasping sound, whistling at times, having its greatest intensity at the junction of the second right costal cartilage with the sternum, transmitted along the vessels; the murmur may sometimes be heard a short distance from the patient. Usually, aortic stenosis is associated with more or less aortic regurgitation, whence a double murmur occurs, having its greatest intensity at the base of the heart, the so-called to and fro, or see-saw murmur.

Prognosis.—So long as compensation is maintained the condition of the patient is comfortable, if a quiet life be followed. When the compensation is ruptured, the usual symptoms of dilatation, venous stasis, and dropsy soon ensue.

TRICUSPID OBSTRUCTION.

This conditions is one of the rarest affections of the heart, and if it ever does occur with or following an attack of endocarditis, the anatomical changes are similar to those of mitral obstruction. It produces enlargement of the heart transversely and is indicated by a presystolic murmur at the base of the ensiform cartilage. This condition soon leads to auricular dilatation; venous stasis rapidly supervenes, associated with venous pulsations similar to those described when speaking of tricuspid regurgitation.

PULMONIC OBSTRUCTION.

Pathologic Anatomy.—Pulmonic obstruction is always a congenital malady and may be found associated with constriction of the pulmonary artery, patulous foramen ovale, patulous ductus Botalli, or stricture of the ductus Botalli. Hypertrophy of the right ventricle may ensue. Those in whom these congenital cardiac conditions occur are otherwise weak, develop slowly, have flabby tissues and soft bones, and seem poorly nourished.

Symptoms.—The hypertrophy which often occurs serves to establish compensation, failure of which as in other valvular defects results in cough, dyspnea, cyanosis, and death. The physical signs reveal marked enlargement of the right ventricle and a systolic murmur in the second left intercostal space which is not transmitted, and a systolic thrill in the pulmonary area.

Prognosis.—The duration of these congenital affections is short, usually from a few days to a few months; although several well-authenticated cases record a much longer duration.

DIAGNOSIS OF VALVULAR DISEASES.

In making a differential diagnosis between the various forms of valvular disease of the heart, strict attention must be paid to the points of greatest intensity at which the several murmurs are heard.

A murmur occurring with or taking the place of the first sound of the heart—the ventricular systole—heard most distinctly at the apex, transmitted to the left axilla and to the inferior angle of the scapula—*a mitral systolic murmur*—signifies *mitral regurgitation.*

A murmur occurring with or taking the place of the first sound of the heart, with its point of greatest intensity at the xyphoid appendix—*a tricuspid systolic murmur*—signifies *tricuspid regurgitation.*

A murmur heard with the first sound of the heart, high-pitched, rasping, or grating in character, with its point of greatest intensity at the second right costal cartilage—*an aortic systolic murmur*—signifies *aortic obstruction.*

A murmur heard with the first sound of the heart, soft in character, with its point of greatest intensity at the junction of the third left costal cartilage with the sternum—*a pulmonic systolic murmur*—signifies *pulmonic obstruction.*

A murmur occuring immediately after the second sound and immediately before the beginning of the first sound of the heart—*a presystolic mitral murmur*—signifies *mitral obstruction.*

A murmur heard with or taking the place of the second sound of the heart, most distinct at the second costal cartilage, to the right of the sternum, and well transmitted toward the apex or below—*an aortic diastolic murmur*—signifies *aortic regurgitation.*

Although eight distinct valvular murmurs have been described as occurring in the heart, those on the right side are of rare occurrence, and hence of little clinical importance.

If a murmur be heard with the *first sound* of the heart, it is almost certainly *aortic obstructive or mitral regurgitant;* and if heard with the *second* sound, it is probably *aortic regurgitant.* A *presystolic mitral* murmur is also of comparatively rare occurrence, the force with which the blood passes from the left auricle into the left ventricle being, under ordinary circumstances, insufficient to excite sonorous vibrations.

Functional or *anemic murmurs* may be confounded with the various forms of valvular disease of the heart. The chief points of distinction between them are, that an anemic murmur, which is always heard at the base of the heart, is always systolic in time, not transmitted away from the heart, and is soft in character, low in pitch, and of variable intensity, now being heard, now entirely absent.

Treatment.—There is no special treatment for each individual form of valvular heart disease. According to Da Costa, the following should govern the treatment:

1. The state of the heart-muscle and of the cavities. 2. The rhythm of the heart-action. 3. The condition of the arteries and veins and of the capillary system. 4. The probable length of existence of the malady and its likely cause. 5. The general health. 6. The secondary results of the cardiac affection.

For practical purposes, it may be considered that if the apex-beat is not displaced, cardiac dullness is not enlarged to the right of the sternum, and dyspnea is not present, medication is not indicated and may even be injurious. If on the other hand, symptoms of hypertrophy, dilatation, or failure of compensation of the heart are present, treatment should be instituted at once.

In all cases, however, the patient should be warned against excessive physical exertion such as rapid walking or running, acsending stairs quickly, excessive work, etc., extremes of passion, exposure to cold and wet, and irregular living. The occurrence of acute diseases in the course of valvular defects cause them to become serious affections and every means should be taken to prevent them. The Nauheim treatment, Oertel's method, and Swedish movements may be of great value during the compensation stage.

The special therapeutic measures indicated for cardiac hypertrophy and cardiac dilatation are considered under those conditions. If the hypertrophy necessary to maintain compensation becomes excessive, aconite, veratrum viride, or nitroglycerin is indicated. If dilatation has occurred, the heart's action is weak and feeble, the circulation is impeded, and venous stasis has followed, digitalis, caffein, strophanthus, and spartein together with more or less active purgation are indicated. Rest is of value in all cardiac conditions. When compensation fails, rest is an absolute indication. The diet should be liquid preferably milk

x Inject sterile Camph Oil m X every 2 hours
alternating hourly with Sol. Adrenalin Chlorde
(1-1000) and m X-XX.

as the passive congestion of the entire digestive tract interferes greatly with assimilation. The heart-balance is best restored by the administration of some preparation of digitalis such as the infusion, ℥i-℥iv (4 to 15 Cc.), the tincture, ♏v to xxx (0.31 to 1.85 Cc.), or the powder, gr. i (0.065 Gm.), three times daily. The possibility of nausea following the use of digitalis, especially the tincture, should be remembered. The dose of the drug is best guided by the results it produces. When for any reason digitalis is not applicable, strophanthus, strychnin, caffein, and spartein may be given. The venous engorgement and dropsy may be relieved by the administration of small doses of mercury and saline purgatives. The combination of calomel, digitalis, and squill, of each gr. i (0.065 Gm.), is especially valuable in this connection. When the dropsy is extreme, tapping or multiple incisions may be required. The extreme and distressing shortness of breath is best relieved by morphin, gr. ¼ (0.0165 Gm.), and inhalations of oxygen. The various coal-tar hypnotics may be employed but are less efficient. When the cardiac rhythm is disturbed from any cause, tincture of belladonna, ♏v to xv (0.31 to 0.92 Cc.), a belladonna plaster over the precordium, or nitroglycerin, gr. 1-100 (0.00065 Gm.), should be employed in addition to other treatment. Sudden failure of the heart requires the x prompt administration of diffusible stimulants such as aromatic spirits of ammonia, whiskey, nitroglycerin, ether, etc. Ammonia and nitrite of amyl should be inhaled while the other remedies should be given hypodermically.

In all cases in which the general health fails and weakness and emaciation present themselves, resort should be had to tonics such as iron, quinin, and arsenic in addition to the other measures.

CARDIAC HYPERTROPHY.

Definition.—An overgrowth or increase in the muscular tissue which forms the walls of the heart, characterized by forcible impulse, over-fullness of the arteries, diminished blood in the veins, and accelerated circulation.

Causes.—It is most frequently caused by obstruction to the outflow of blood such as results from valvular disease of the heart, emphysema, Bright's disease, and arteriosclerosis but it may also be due to excessive functional activity, such as produced by prolonged muscular exertion, exophthalmic goiter, the long-continued use of large amounts of tea, coffee, and tobacco, and attempts to overcome pericardial adhesions.

Varieties.—I. *Simple hypertrophy*, or a simple increase in the thickness of the cardiac walls; II. *Eccentric hypertrophy*, increase in the cardiac walls and dilatation of the cavities, causing a *dilated hypertrophy;* III. *Concentric hypertrophy,* increase in the cardiac walls with decrease of the cavities, a very rare form.

Pathologic Anatomy.—Hypertrophy of the heart is usually limited to the left side, the ventricles being more commonly involved than the auricles, the latter dilating. The shape of the heart is altered by hypertrophy; if the right ventricle, the heart is widened transversely and the apex blunted; if the left ventricle, the heart is elongated and, as a rule, the cavity is dilated; if both ventricles are hypertrophied, the heart has a globular shape. From increase in weight the heart may fall back during the recumbent position, thereby lessening the area of cardiac dullness, but during the sitting or upright posture it sinks lower in the chest and to the left, causing more or less prominence of the abdomen. The increase in the size of the organ is a true increase or hypertrophy of the muscular tissue, and not a hyperplasia. The tissue is firmer and the color brighter and fresher than when the size of the organ is normal. The *cor bovinum* of the old writers is an enormous hypertrophy of the heart with dilatation of its cavities.

Symptoms.—These depend upon the amount of hypertrophy. If only sufficient to compensate for valvular defects or other circulatory disturbances there will be no symptoms. When the enlargement is disproportionate to the obstruction, it is manifested by increased and forcible cardiac action, precordial discomfort, headache, dizziness, ringing in the ears, flushes or flashes of light, dyspnea on exertion, congestion of the face and eyes, dry cough, epistaxis, and restless nights, with more or less jerking of the limbs. The arteries become full and the pulse is firm, and bounding. The carotids and superficial arteries pulsate markedly, the patient frequently complaining of throbbing sensations. A sphygmographic tracing shows the line of ascent vertical and abrupt, but the apex is rounded, and the line of descent is oblique, unless there is more or less insufficiency of the valves.

Physical Signs.—*Inspection* reveals fullness or prominence of the precordium with a distinct impulse.

Palpation detects the impulse one or two intercostal spaces lower down and to the left. It is stronger and more or less diffused—the heaving impulse.

Percussion determines an increase in the area of cardiac dullness vertically and transversely on the left side of the sternum, unless the right ventricle is also hypertrophied, when the cardiac dullness is increased to the right of the sternum.

Auscultation in simple hypertrophy without any valvular changes, detects a loud first sound of a somewhat metallic quality, the second sound being strongly accentuated. In the presence of valvular disease the characteristic murmurs are heard in addition.

Sequels.—Cerebral hemorrhage, miliary cerebral aneurysms, cardiac dilatation, and fatty degeneration may be mentioned as the most common sequels.

Diagnosis.—The history, course, symptoms, and physical signs are distinctive and when carefully considered should prevent error in diagnosis.

Prognosis.—When it is the result of valvular disease, the hypertrophy is said to be compensatory. If the result of Bright's disease, emphysema of the lung, or if occurring late in life, or associated with atheromatous degeneration of the vessels, the prognosis is unfavorable. When it is the result of functional over-action in the strong and robust, a further enlargement can often be prevented by active and persistent treatment.

Treatment.—When the hypertrophy is excessive, the indications are to remove the cause if possible and to lessen the force and number of the cardiac pulsations.

The habits of the patient should be corrected; all laborious or active exertion should be restricted and the recumbent posture should be assumed several hours during the day, if possible. The diet should be regulated, and all forms of stimulants such as liquors, tobacco, tea, and coffee should be interdicted. Cases of cardiac hypertrophy associated with Bright's disease are often relieved by digitalis. In rare instances cardiac pain follows the use of digitalis; in such cases, caffein citrate or strophantus are to be employed. When the hypertrophy is associated with anemia, iron should be administered in addition to other measures.

The force and frequency of the cardiac pulsations are best controlled by the long-continued use of tincture of aconite, ♏i-ii (0.06-0.21 Cc.), three times daily, or tincture of veratum viride, ♏i-ii (0.06-0.12 Cc.), three times daily, together with the administration of saline purgatives, bromids, and nitrites to lessen the arterial tension and to relieve the symptoms.

DILATATION OF THE HEART.

Definition.—An increase in the size of one or more of the cavities of the heart, characterized by feebleness of the circulation, terminating in venous stasis, cyanosis, edema, and exhaustion.

Causes.—It is usually brought about by chronic valvular heart-disease, emphysema, chronic bronchitis, gout, Bright's disease, alcoholism, or syphilis, but may be due to over-exertion in those of feeble resisting powers, such as youths and soldiers.

Varieties.—I. *Simple dilatation*, the cavities being enlarged, the walls normal. II. *Active dilatation*, corresponding to eccentric hypertrophy; the cavities being enlarged and the walls increased in thickness, the so-called "dilated hypertrophy." III. *Passive dilatation*, the cavities being enlarged and the walls thinned or stretched.

Pathologic Anatomy.—The right side of the heart is far more frequently involved than the left side. The shape of the organ is altered, depending on the part affected. The weight of the organ is, as a rule, increased, as hypertrophy almost always accompanies or precedes dilatation. The muscular tissue is generally pale, mottled, and softened, and under the microscope presents evidences of degeneration. The orifices also participate, and especially the auriculo-ventricular orifice, resulting in the valves becoming incompetent to close the orifices, and this latter effect is increased by the removal of the basis of the papillary muscles a greater distance from the orifice, in consequence of the distension of the wall. When the auricles dilate, the large venous trunks opening into them, unprotected by valves, commonly participate in the dilatation, and may become greatly enlarged. The passive congestion of the organs that follows the enfeeblement of the circulation produces changes in their structure.

Symptoms.—The manifestations are referable to the enfeebled circulation and include feeble pulse, headache aggravated by the upright position, attacks of syncope, cough, dyspnea, jaundice, dyspepsia, constipation, scanty, often albuminous urine, mental dullness, vertigo, often relieved by a copious epistaxis, and finally dropsy beginning in the lower extremities. The condition terminates in death by exhaustion. Treatment may serve to temporarily relieve any of the symptoms just mentioned.

Physical Signs.—*Inspection* detects enlargement and distention of the superficial veins and an indistinct, often wavy and diffused, cardiac impulse. If tricuspid regurgitation is present, jugular pulsation will be observed.

Palpation confirms inspection; the impulse is feeble, irregular, and heaving.

Percussion serves to determine extension of the area of cardiac dullness transversely and especially toward the right side.

Auscultation in the presence of valvular lesions reveals characteristic murmurs. If there are no valvular lesions, the cardiac sounds are weaker than normal and the first sound is sharper in quality than usual.

Diagnosis.—*Hypertrophy* of the heart shows increased cardiac dullness, and is a disease of powerful cardiac action, while dilatation is an affection of feeble action associated with dropsy.

Pericardial effusion has many points of resemblance to cardiac dilatation, but it begins suddenly, associated with some acute malady, and while the heart sounds are indistinct or feeble at the apex, they both have their normal qualities at the cardiac base, while dilatation of the heart has a chronic history, and results in general venous stasis, the cardiac sounds being of the same intensity over the entire precordium.

Prognosis.—The outlook is unfavorable. Death results gradually from exhaustion or suddenly from cardiac paralysis induced by some undue excitement.

Treatment.—Dilatation of the heart is incurable. The symptoms may, however, be temporarily relieved and the course prolonged. In all cases the indications are to improve and maintain the general nutrition of the patient and to control and steady the cardiac action.

The first indication is met by a generous diet, moderate exercise, and the administration of stomachics, red wine, iron, etc.

The second indication is met by strict observance of the rules of hygiene, by moderate exercise, and by the administration of heart tonics such as digitalis. This drug may be used in the form of powder, tincture, or infusion or in the following combination:

℞. Tincturæ nucis vomicæ	f℥ ss	15. Cc.
Tincturæ digitalis	f℥ ss	15. Cc.

M. S.—Fifteen drops after meals, in water.

The combination of tincture of strophanthus with digitalis is very beneficial. Strychnin sulphate, gr. 1-24 (0.0025 Gm.) three times daily, caffein citrate, gr. i-iii (0.06-0.2 Gm.), three times daily, and spartein sulphate, gr. ⅛-i (0.0081-0.065 Gm.), three times daily, are also valuable heart tonics and stimulants. Morphin sulphate, in small doses hypodermically, often acts like magic in restoring the circulation (Bartholow) especially when compensation is failing, and dropsy and cyanosis become marked.

The following pill is often of great advantage:

℞. Ferri reduct.	gr. j-ij	.065-.13 Gm.
Quininæ sulph.	gr. j-ij	.065-.13 Gm.
Pulv. digitalis	gr. j	.065 Gm.
Morphinæ sulph.	gr. 1-24	.0025 Gm.

M. S.—Three times a day

An excellent combination is the following:

℞. Tinct. digitalis	fʒ iss	6. Cc.
Tinct. cacti grandiflor. ..	f℥ j	30. Cc.
Caffeinæ citratis	ʒ j	4. Gm.
Tinct. card. comp. ..ad	f℥ iv	ad 120. Cc.

M. S.—Teaspoonful, diluted, three or four times daily.

The bowels, skin, and kidneys should be kept in action, using, if needed, purgatives, diaphoretics, and diuretics. The following combination, suggested by Dr. J. M. Anders, is satisfactory in many instances:

℞.	Caffeinæ citratis	ʒ j	4. Gm.
	Strychninæ sulph.	gr. ⅓	.022 Gm.
	Sparteinæ sulph.	gr. ij	.13 Gm.

M.—Ft. capsulæ No. xij.
S.—One every three or four hours.

Or the following excellent diuretic pill:

℞.	Pulv. scillæ	gr. xxx	2. Gm.
	Pulv. digitalis	gr. xxx	2. Gm.
	Caffeinæ citratis	gr. xxx	2. Gm.
	Hydrarg. chlor. mitis	gr. v	.3 Gm.

M.—Ft. pilulæ No. xxx.
S.—One three or four times daily.

The development of pulmonary congestion calls for the use of dry cups, digitalis, caffein, atropin, and stimulants. For hepatic congestion, blue mass and popophyllin are indicated. Cardiac asthma may be relieved to a great extent by dry cups, morphin (hypodermically), or Hoffman's anodyne. The dropsy may be lessened by dry cups over the kidneys, digitalis, potassium acetate, scoparius, preparations of juniper berries, and compound jalap powder. If the dropsy is uninfluenced by these means, calomel, gr. iii (0.2 Gm.), gaurded by powdered opium, gr. 1-12 (0.005 Gm.), three times daily, should be employed.

The treatment of cardiac dilatation and cardiac failure by baths and systematic exercise has excited much interest and discussion recently, with the result of its indorsement in certain cases. Exercise is employed in one of three methods or, rarely, a combination of these: (1) passive exercise and massage (Swedish or Ling plan; (2) movements with limited resistance (Schott plan, but really a modification of the Swedish); (3) method of climbing (Oertel). The baths are those of the Nauheim (saline) natural waters. A number of American and English clinicians report good results with artificial Nauheim baths. This system of cardiac treatment is combined with regulated diet, business rest, and the use of some cardiac tonics.

ACUTE MYOCARDITIS.

Synonyms.—Carditis; abscess of the heart.

Definition.—An inflammation of the muscular tissue of the heart, characterized by pain, feeble circulation, symptoms of blood-poisoning, and collapse.

Causes.—It nearly always arises as the result of some general septic condition such as pyemia, septicemia, typhoid fever, puerperal fever, etc., but it may be due to extension from a septic pericarditis or endocarditis.

Pathologic Anatomy.—The structural changes consist in discoloration and softening of the cardiac substance with infiltration of a sero-sanguineous fluid, fibrinous exudation, and pus, leading ultimately to abscess formation in the myocardium. The affection terminates in either cardiac aneurysm or rupture of the heart. In the event of recovery, depressed cicatrices or scars will be found marking the sites of former abscesses.

Symptoms.—The clinical evidences of inflammation of the cardiac muscles are very vague. If, during the course of one of the maladies mentioned, there are developed precordial pain, irregular and feeble cardiac action, cardiac dyspnea, pyrexia of a low type, with symptoms of blood-poisoning and a tendency to collapse, or the symptoms of the so-called typhoid state, myocarditis may be suspected.

Diagnosis.—The diagnosis is seldom made before death. It may be presumed, however, if in the course of septic conditions, symptoms of heart failure occur.

Prognosis.—The course of acute myocarditis is very rapid, death being the usual termination in from three to five days.

Treatment.—Cardiac stimulants should be employed freely in addition to the other remedial measures indicated by the general sepsis.

CHRONIC MYOCARDITIS.

Synonyms.—Fibroid heart; chronic interstitial myocarditis; fibrous myocarditis; chronic carditis; cardio-sclerosis.

Definition.—A slowly developing hyperplasia of the interstitial connective-tissue of the heart, leading to induration of its substance; characterized by shortness of breath on slight exertion, attacks of tachycardia, precordial pain, disordered circulation, and vertigo. It is proper to state that many cases present no symptoms whatever.

Causes.—The most frequent cause is sclerosis of the coronary arteries, leading to imperfect blood-supply to the cardiac muscles. It usually occurs in the aged and bears a direct relation to the condition of the arteries throughout the body. Among other causes may be mentioned diseases of the kidneys, alcoholism, excessive use of tobacco, syphilis, pericarditis, endocarditis, and acute myocarditis. There is, undoubtedly, often an inherited predisposition to fibroid changes in the vessels, in which case the causes named would act as exciting causes.

Pathologic Anatomy.—The heart is enlarged and dilated and its structural changes may be diffused or limited to the walls of the left ventricle, the papillary muscles, or the septum. Valvular disease may be present. Atheroma is usually present in one or more of the coronary arteries and may involve the aorta. Sudden complete closure of one coronary artery in the course of these morbid changes is usually fatal.

On section of the organ, the cardiac wall will be found to cut with distinct resistance, due to an overgrowth of the interfibrillar connective-tissue and the development of new fibrous tissue. When due to some general intoxication as accompanies gout, syphilis, alcoholism, etc., these changes are uniformly distributed but if due to embolism, thrombosis, or other conditions occluding one or more coronary vessels there will be localized anemic infarction and sclerosis. On microscopic examination, the muscle bundles will be found degenerated and replaced by fibrous tissue. In cases due to syphilis, the terminal branches of the coronary arteries are narrowed and sclerotic to the point of obliteration. The inelastic fibrous tissue in the heart is often insufficient in resisting the intracardial pressure and gives way, resulting in aneurysm of the heart. This is particularly liable to occur in localized cardiosclerosis.

Symptoms.—The great majority of patients having chronic myocarditis present no symptoms until an extra cardiac effort is demanded. An early symptom is breathlessness on slight exertion, with either cardiac palpitation or a feeble, irregular pulse. Vertigo is frequent and distressing, increased by indigestion and constipation. Anginal attacks (cardiac pain) or sensations of constriction or pressure over the precordium are frequent, often following some exertion or an attack of indigestion. The pulse-rate is often decreased in frequency in cases which present no other symptom. A frequent symptom is syncope, coming without warning or after sudden exertion, the result of sudden failure of the cerebral circulation. Among other periodical symptoms are cardiac asthma, pseudo-apoplectic attacks, and hepatic, gastric, and nephritic disorders. As the fibroid changes progress, there develop progressive weakness, dyspnea, insomnia, disordered digestion, and cerebral weakness, often showing itself as mania, delusional attacks, or dementia.

Physical Signs.—*Inspection* and *palpation* recognize a feeble impulse which at times is scarcely appreciable.

Percussion detects enlargement of the area of cardiac dullness due to the dilated hypertrophy of the heart.

Auscultation shows the first sound of the heart to be valvular in quality, the booming or muscular quality having disappeared. Murmurs are frequent and are due to valvular disease. A very characteristic feature is irregularity in the rhythm and force of the heart, a forcible contraction alternating with a weak contraction. Eventually both sounds become weak and feeble.

Diagnosis.—The points of value in arriving at a diagnosis are: a careful study of the first sound of the heart at the apex; the character of murmurs if present, the conditions of the arteries, the dyspnea, the feeble, irregular pulse in patients past fifty years, and the occurrence of anginal attacks after exertion or mental worry.

Prognosis.—This is controlled by the habits of the patient. The disease is incurable, but life may be fairly comfortable for many years if care be exercised. It should be remembered, however, that chronic myocarditis is one of the most common causes of heart-failure and subsequent death, in the course of acute pneumonia, typhoid fever, etc., and after over-exertion of any kind.

Treatment.—No remedy can remove the fibroid change. The indications are to promote the patient's nutrition, hold in check the progress of the fibrosis, and meet or prevent the symptoms as they arise. The patient's general condition requires the administration or iron, arsenic, and the hypophosphites. Constipation should be avoided by the use of aloes, cascara, or other mild laxative. Mental strain and physical exertion should be carefully guarded against and tobacco and malt liquors should be interdicted. The diet must be plain with but little tea or coffee. In the elderly, a small amount of good whiskey once or twice daily is valuable. The Nauheim treatment may be of great benefit and is worthy of a trial.

Relief of the symptoms is usually obtained by measures directed towards supporting the heart. For breathlessness, spirits of glonoin, spirits of nitrous ether, and aromatic spirits of ammonia are especially indicated. Cardiac palpitation may be relieved by potassium bromid, lithium bromid, and aromatic spirits of ammonia. Weakness of the heart requires the administration of strychnin sulphate, gr. 1-24 (0.0025 Gm.), three times daily, tincture of digitalis, ♏x-xx (0.6-1.2 Cc.), three times daily, or caffein citrat, gr. iii (0.2 Gm.), three times daily. The recumbent position should be assumed, and gastrointestinal disturbances should receive prompt treatment. For the anginal attacks, hypodermic injections of morphin sulphate, gr. ⅛-¼ (0.008-0.016 Gm.), or chlorodyne, ♏x-xx (0.6-1.2 Cc.), should be given repeated as needed. When syncopal attacks occur, the patient should be placed in bed and a mustard plaster applied to the precordium; stimulants, especially nitroglycerin, should be administered, preferably by hypodermic injection. The following is an excellent combination for the relief of dyspnea, vertigo, and chest pains:

℞. Lithii bromidi	ʒ vss	22. Gm.
Spiriti glonoini	♏ xvj	1. Cc.
Liq. potassii citratis q. s.	ad f℥ viij	ad 240. Cc.

M. S.—Tablespoonful four times daily, diluted.

FATTY HEART.

Synonyms.—Fatty degeneration of the heart; chronic myocarditis.

Definition.—A change in the muscular fibers of the heart, in which the transverse striæ are replaced by granules and globules of fat, characterized by feeble cardiac action, venous stasis, and dyspnea.

Causes.—The most important factors in the production of this condition are impaired nutrition in the elderly, prolonged anemia, chronic gout, alcoholism, phosphorous poisoning, cancer, tuberculosis, and disease of the coronary arteries.

Pathologic Anatomy.—Fatty degeneration should be distinguished from fatty infiltration; in the latter, the adipose tissue is deposited on the organ and between its muscular fibers. This condition is to some extent normal and accompanies general obesity.

Fatty degeneration affects the individual muscle fibers; the changes being within and not between the fibers. The fatty metamorphosis may affect the whole organ, or the entire ventricular walls, or may be limited to portions of them. If the degeneration be marked, the color is yellowish, the tissues soft and easily torn, and to the touch have a greasy feeling, oil being yielded on pressure.

The microscopic changes are characteristic. The striæ of the muscle are rendered indistinct by fat and oil globules, gradually becoming more and more obscured, and finally disappearing altogether, the fibers being replaced by fat granules.

Symptoms.—The manifestations of fatty degeneration are weakness of the heart, anemia of the various organs, and venous stasis. The cardiac action is slow, feeble and irregular and the pulse is compressible. Precordial distress is present, often aggravated by attacks of angina pectoris. Dyspnea, increased on exertion is also a symptom. Anemia of the brain induces vertigo, swooning, and pseudoepileptic attacks especially marked on suddenly rising from a recumbent posture. Anemia of the lungs gives rise to a dry hacking cough. Anemia of the gastrointestinal tract produces dyspepsia and constipation. Renal anemia is followed by scanty, often albuminous, urine and dropsy beginning in the lower extremities. Weakness and pallor are common symptoms.

A formidable symptom, causing much inconvenience as well as alarm to the patient, is that which he will term his constant " sighing," the Cheyne-Stokes breathing—"A pause in the breathing, a complete suspension of the respiratory acts for a period of time (during which breathing might occur several times in the normal manner), then the resumption of respiration very feebly and slowly, and a gradual and progressive increase in the number and depth of respirations until the maximum is reached, and then again a gradual and progressive diminution in the same order, in the number and depth of the respirations, until another pause occurs"—the "oscillating respiration."

Concomitant symptoms are atheromatous changes in the vessels, and the *arcus senilis.*

Physical Signs.—*Palpation* detects a weak and irregular cardiac impulse.

Percussion determines no change in the area of cardiac dullness unless cardiac hypertrophy is present.

Auscultation reveals a feeble, toneless, almost inaudible first sound. The second sound is normal. Murmurs are not present unless there are coincident valvular lesions.

Diagnosis.—Feeble cardiac sounds, with slow pulse, attacks of cardiac asthma or Cheyne-Stokes breathing, and evidences of arcus senilis, make the diagnosis very certain. The question of fibroid heart must always be considered.

Prognosis.—The outlook is unfavorable. Life may be prolonged by appropriate treatment but death is liable to occur at any time from cardiac paralysis, rupture of the heart, or exhaustion.

Treatment.—There is no treatment capable of restoring the degenerated muscle-fibers to their normal condition. Various means may be employed, however, for lessening the severity of the symptoms. Mental and physical exertion should be avoided. The diet should be generous and consist of easily digested substances. Moderate exercise should be prescribed. Stimulants such as iron, quinin, strychnin, cod-liver oil, and hypophosphites should be administered over an indefinite period to strengthen and maintain the body-tone. All the secretions should be kept active to relieve the crippled heart from any unnecessary strain. The recumbent posture should be assumed for several hours each day. The Nauheim treatment is applicable. The heart's action is best sustained by strychnin sulphate, gr. 1-48 - 1-32 (0.0015-0.002 Gm.), three or four times daily, but caffein, spartein, and nux vomica may also be employed. Digitalis is contraindicated in advanced cases. For syncopal attacks, nitroglycerin, spirits of nitrous ether, aromatic spirits of ammonia, and hypodermic injections of ether, camphor, or whiskey are indicated.

PALPITATION OF THE HEART.

Synonym.—Irritable heart.

Definition.—A functional disturbance of the heart; characterized by increasing frequency of its movements and more or less irregularity of the rhythm, with a strong tendency toward hypertrophy.

Causes.—Among the more important causes may be mentioned female sex, puberty, menstrual disorders, anemia, emotion, mental anxiety, hysteria, over-exertion, exhaustion following acute or chronic disease, "heart-strain" (Da Costa), dyspepsia, long-continued use of tea, coffee, tobacco, and alcohol in large quantities, and excessive venery.

Symptoms.—Usually palpitation of the heart has a sudden onset after some one of the causes mentioned, with precordial oppression or pain; rapid, tumultuous beating, the impulse being visible through the patient's clothing; dyspnea, anxiety, and a sense of choking or fullness in the throat, the recumbent position being impossible; vertigo, faintness, flashes of light, the pulse full and strong or feeble, and the face flushed or pale, the patient having a feeling of anxiety with a sense of impending danger and a fear of sudden death. These attacks are paroxysmal, lasting from a few moments to several hours, or a day, the patient often voiding a large quantity of limpid urine after the paroxysm has subsided, when there is a strong tendency to sleep.

Diagnosis.—Irritability of the heart is differentiated from the various forms of cardiac disease by the absence of all the physical signs mentioned as occurring in those conditions.

Prognosis.—If early and properly treated, favorable.

Treatment.—The first point in the treatment of irritability of the heart is to remove the cause; the next, to prevent the recurrence of the attacks of palpitation.

The majority of cases do well after a few doses of either compound spirits of ether (Hoffman's anodyne) or aromatic spirits of ammonia, or a combination of digitalis and belladonna. Permanent relief is often afforded by a combination of potassium bromid and veratrum viride. Trional, gr. x-xv (0.6-1 Gm.), three times daily, is often useful. If the patient be anemic, excellent results follow the prolonged use of the elixir of iron, quinin, and strychnin. Locally, belladonna plaster to the precordium affords relief. The acute attack is often wonderfully benefited by ice over the precordium.

TACHYCARDIA.

Synonyms.—Rapid heart; quick heart; paroxysmal rapid heart.

Definition.—Paroxysmal rapid cardiac action, with or without subjective symptoms.

Causes.—The direct cause is somewhat obscure. The condition may be found associated with one of the crises of cerebral or spinal disease, the menopause, neuritis of the pneumogastric nerve, chronic myocarditis, neurasthenia, chronic gastritis, the excessive use of tobacco, petit mal, pyrexia, lesions of the base of the brain, etc.

Pathology.—The affection has no structural lesions peculiar to itself. There may be paralysis of the inhibitory fibers of the vagus, a direct irritation of the accelerators of the sympathetic, or reflex irritation from some lesion in the cardiac wall or elsewhere in the body.

Symptoms.—The paroxysm is sudden in its onset, with or without "warnings"—if these latter occur, they are in the shape of vertigo, ringing in the ears, and a sense of impending danger. The cardiac action is increased to 150, 175, 200, rarely 250 beats per minute. The pulse is small, weak, easily compressible, and often irregular, with carotid pulsation (which indicates emptiness and low tension of the artery, as in aortic regurgitation). The respiration is slightly increased; rarely there is dyspnea. The surface is at first pale, but soon becomes flushed. The expression is anxious and denotes suffering. There is a feeling of precordial constriction, with more or less smothering. Rarely, subjective symptoms are absent.

The duration is from a few minutes to hours, or days. The attack usually ceases during sleep, but the rapidity of the pulse may continue during the disturbed sleep.

Auscultation detects a clear and ringing first sound, the strong and booming character being absent. The second sound is weak and lacks the valvular quality of the normal. A murmur is often heard at the apex.

Diagnosis.—The principal points in distinguishing tachycardia from other cardiac affections are the paroxysmal character, and the great increase in the pulse-rate and cardiac action of which the patient may or may not be conscious.

Prognosis.—When occurring as a pure neurosis or as the result of some cause that permits of easy removal, the prognosis is good. It is often, however, an unfavorable symptom of some central lesion. When it develops in persons suffering from chronic myocarditis or atheroma of the vessels, it is liable to terminate suddenly in death.

Treatment.—As in other affections involving the cardiac functions, rest in bed is of great importance in the treatment. The application of ice to the precordium, together with the hypodermic injection of morphin sulphate, gr. 1/6 (0.011 Gm.), and atropin sulphate, gr. 1-100 (0.00065 Gm.), are of great benefit during the paroxysm. Occasionally, the administration of a few large doses of digitalis brings about the restoration of cardiac equilibrium. Sedatives such as tincture of belladonna, the bromids, camphor, trional, etc., are at times valuable in arresting the attacks. Among other valuable measures may be mentioned compression of the vagus in the neck, the taking of a full and deep inspiration, and Rosenfeld's method in which the patient is directed to squeeze his arms and elbows into his sides and at the same time forcibly compress his abdomen. During the intervals between the paroxysms, the habits should be regulated and harmful substances, such as alcohol, tobacco, tea, and coffee should be interdicted.

BRADYCARDIA.

Synonym.—Brachycardia.

Definition.—A paroxysmal or permanent slowness in the cardiac action. It is agreed that bradycardia begins when the pulse is reduced to at least forty beats per minute.

Causes.—It is often associated with organic nervous diseases and is a symptom of such cardiac diseases, as fibroid and fatty heart and atheroma of the coronary arteries.

The condition frequently occurs during convalescence from infectious diseases, such as diphtheria, pneumonia, typhoid fever, erysipelas, and rheumatism; uremia, lead-poisoning, anemia, and chronic alcoholism are often causes. According to Balfour, "Many, if not most, of the sufferers from bradycardia are epileptics."

Symptoms.—The slow action of the heart, varying from 40 to 8 beats per minute, is the most prominent manifestation. The pulse is weak, small, and slow. The first sound of the heart is soft and feeble and often the second sound is inaudible. As a result of the slow cardiac action there are noises in the ears, vertigo, syncopal attacks, and rarely convulsions. Premonitory signs may or may not be present.

Prognosis.—The outlook depends entirely upon the cause. When due to grave organic disease, sudden death is not an uncommon termination.

Treatment.—As long as the slow cardiac pulsations are sufficient to supply the requirements of the economy, medication is not needed; when, however, the reverse obtains, rest in the recumbent position, heat to the precordium, and the use of such remedies as atropin sulphate, caffein citrate, strychnin sulphate, spirits of glonoin, and aromatic spirits of ammonia are indicated. Often the emergency is so great as to call for the hypodermic use of the selected drug.

Digitalis is contraindicated. Between the paroxysms, such remedies as improve the general health and prevent the progress of the central or exciting cause are required.

ARHYTHMIA.

Synonyms.—Arhythmia cordis; irregularity of the pulse.

Definition.—A lack of cardiac rhythm, or irregularity in the cardiac pulsations. It is a symptom rather than a disease.

Causes.—Valvular diseases; myocardial diseases; cardiac dilated hypertrophy; atheroma of coronary arteries and aorta; excessive use of tobacco, tea, or coffee; flatulent dyspepsia; neurasthenia, hysteria, and melancholia.

Symptoms.—An irregularity in cardiac action, either in the rhythm or the regularity of the force of the beats, or an intermission in the cardiac contractions. Symptoms referable to the underlying cause are also present.

Diagnosis.—An examination of the pulse, auscultation of the heart, and the use of the sphygmograph determine the arhythmia.

Prognosis.—Depends upon the cause. In functional cases favorable, in organic cases unfavorable.

Treatment.—In purely functional conditions rest of mind and body with regulation of the diet, attention to the secretions, and the administration of the bromids are of great value. In other cases, strychnin or digitalis in addition to treatment directed towards the underlying cause is indicated.

ANGINA PECTORIS.

Synonyms.—Neuralgia of the heart; breast-pang.

Definition.—Paroxysms in which there occur sharp cardiac pains, extending usually into the left shoulder and down the left arm, accompanied by a feeling of constriction of the thorax and a strong fear of impending death.

Causes.—The direct cause of the affection is insufficient nutrition of the heart. This deficiency may be brought about by disease or obstruction of the coronary arteries, diseased conditions of the aortic valve, pressure of an adjacent tumor, excessive dilatation or enlargement of the heart, adhesive pericarditis, habitual use of tobacco, etc. The tendency may be inherited. Syphilis and hysteria may exert an influence in its production. According to Trousseau it may be considered as a form of masked epilepsy or it may alternate with true epileptic attacks. Allbutt believes it to be due to an acute aortitis. Male adults after 40 years of age are most often affected and the attacks are precipitated by over-exertion, great mental excitement, or acute indigestion.

Pathology.—The most constant structural changes are sclerosis, atheroma, and obliteration of the coronary arteries. Such changes may be present without angina and, on the other hand, anginoid attacks may occur independent of structural alterations. Functional disturbances of the cardiac plexuses are responsible for the symptoms.

Symptoms.—The chief symptom is intense agonizing pain which begins in the region of the heart and extends to the neck and down the left arm. Shortness of breath, and precordial oppression are present. The chest is fixed and the heart's action is weak and feeble. The face is pale or ashen gray, the expression is anxious, and there is a fearful sense of impending death. The body and face are covered with drops of cold sweat. The pain usually lasts but a few seconds or minutes. Extreme prostration follows the attack unless it is terminated meanwhile by death. The end of the paroxysm is marked by cessation of the pain and precordial distress, and by vomiting, or excessive flow of urine. The first attack may end in death or there may be a recurrence at varying periods, soemtimes extending over years.

The unpleasant sensations of these patients during an attack, and the nervous disorder associated with it, slowly bring about a mental change. They are depressed and gloomy, sometimes suicidal, and often develop epilepsy.

Attacks of angina in nervous women and children, the hysteric or pseudo-anginal attacks, come on gradually with distention of the abdomen, eructations of gases, excessive restlessness, flushed face, irritable pulse, diffused precordial pain, and general hysteric phenomena.

In a few cases the pain is absent, but all other symptoms are present—the "*angina sine dolore*" of Gairdner. Balfour claims that pain is not an essential part of the disease.

Diagnosis.—The points to be remembered are that the attacks are always paroxysmal, with long or short intervals, the patient having a sense of coldness, and frequently a cold sweat, the heart's action being not increased, the chest fixed, and the breathing slow. *Intercostal neuralgia* and *gastralgia* may be confused with this affection but the history and concomitant symptoms of the former conditions will aid greatly in making a diagnosis. These affections lack the characteristic paroxysms observed in true angina pectoris.

Prognosis.—The outlook in true angina pectoris is unfavorable. Seventy-five percent recover from the first attack but the affection ultimately terminates fatally. In pseudo-angina the prognosis is always favorable.

Treatment.—*The Attack:* Prompt relief follows the immediate inhalation of amyl nitrite, ♏iii-v (0.2-0.3 Cc.), or chloroform, or the hypodermic injection of morphin sulphate, gr. ¼-½ (0.016-0.032 Gm.), combined with atropin sulphate, gr. 1-100 (0.00065 Gm.), or nitroglycerin, gr. 1-100 - 1-50 (0.00065-0.0013 Gm.). In many cases, the use of nitroglycerin, gr. 1-100 (0.00065 Gm.), three times daily, over an extended period, lessens not only the frequency but also the severity of the paroxysm. Chlorodyne, ♏x-xv (0.6-1 Cc.), repeated often affords relief. Spartein sulphate, gr. ¼ (0.016 Gm.), three times daily, is also highly recommended. The application of a mustard plaster or other forms of counterirritation to the precordium is productive of considerable benefit.

The Interval: Attempts should be made to remove the exciting causes or to diminish their influence. Great care should be exercised in the diet that flatulency and constipation do not occur. Mental excitement and physical exertion should be avoided. When structural changes are suspected, potassium iodid, gr. x-xx (0.6-1.3 Gm.), three times daily, should be administered. The nitrites, and nitroglycerin should be employed as they lessen materially the frequency and severity of the paroxysms. Tonics such as iron, arsenic, strychnin, phosphorus, etc., are of value in that they increase the resistance of the body and improve its general tone. Strophanthus and strychnin are indicated when the heart is weak. Trousseau advises the long-continued administration of small doses of belladonna. Quain employs the constant current, applying the positive pole over the sternum and the negative pole over the lower vertebræ. The Nauheim treatment, particularly the hot baths, may be of benefit. The *cold baths are positively harmful* in this condition.

Pseudoangina requires the treatment prescribed for hysteria in general.

ARTERIOSCLEROSIS.

Synonyms. — Atheroma; arteriocapillary fibrosis (Gull and Sutton); endarteritis chronica deformans (Virchow).

Definition.—A chronic degenerative and inflammatory disease of the vascular system, resulting in an overgrowth of the connective tissues of the arteries, followed by calcareous deposits. The changes may extend to the capillaries and veins. As a result of the impairment of the arterial circulation, there occur fibroid degenerations in other organs, resulting in loss of elasticity in the walls of the vessels, increase of arterial tension, narrowing of the caliber of smaller arteries, and impairment of the nutrition of the organs supplied.

Causes.—The principal etiologic factors are senility, heredity, male sex, alcoholism, syphilis, lead-poisoning, diabetes, malaria, gout, rheumatism, lithemia, Bright's disease, exposure, and excesses of various kinds. The condition of the arteriocapillary system may be taken as an index of an individual's age.

Pathologic Anatomy.—The atheromatous changes are most frequent in the aorta. Rokitansky gives the relative order in which atheromatous degenerations occur as follows: aorta, splenic, femoral, iliac, coronary arteries of the heart, arteries of the brain, uterine, subclavian, brachial, ulnar, and radial.

The internal surface of the affected vessels is irregularly thickened with gelatinous and translucent, dense and fibrous, or calcareous deposits. If the calcification is extensive, the vessel is changed into a hard, stiff tube. Often the surface of the thickening or deposit is destroyed, presenting the so-called "atheromatous ulcers," which may be covered with masses of thrombi.

The above conditions are the result of inflammatory change in the intima of the affected vessel which appears three or four times as thick as normal, due to the swelling of its elements, the new

"A man is as old as his arteries."

growth of connective-tissue, and the deposit of round cells. Fatty degeneration of the inflammatory products is the common sequence.

The result of the changes in the arteries is a loss of elasticity, thus hindering the propulsion of the blood-current and raising the arterial tension, ultimately leading to hypertrophy of the left ventricle. These changes finally involve the coronary arteries and lead to alterations in the myocardium. The nutrition of various other organs is likewise impaired when the intima of their respective arteries is involved in the degenerative process.

Symptoms.—These are not always apparent and vary with the arteries involved and their distribution. When the process is general, the peripheral arteries have a hard bony feel, not unlike whip-cord. The increased resistance of the arterial system induces increased cardiac activity and consequent hypertrophy.

Attacks of vertigo, pseudo-apoplectic attacks, or spells of unconsciousness in the aged or those having superficial hardened arteries, are generally due to changes in the cerebral vessels. Evidences of myocarditis and angina pectoris point to atheroma of the aorta and coronary arteries. Renal arteriosclerosis manifests itself as chronic interstitial nephritis. Gangrene of the extremities in the old—senile gangrene—point to atheroma or thrombi, the result of the fibrosis.

Physical Signs.—*Palpation* reveals a forcible cardiac impulse in the early stages. The superficial arteries are hard and those at the wrists feel like a string of beads pulsating.

Percussion shows increased precordial dullness especially over the left ventricle.

Auscultation in the early stage detects prolongation of the first sound with accentuation of the second sound over the aortic cartilage. As the heart dilates and the walls become diseased, the sound becomes feeble and often irregular and intermittent.

Sequels.—As consequences of this condition of the arterial system may be mentioned cerebral hemorrhage, thrombosis, embolism, or aneurysm, myocarditis, angina pectoris, chronic interstitial nephritis, gangrene, aneurysm, etc.

Prognosis.—The structural changes may be prevented or retarded but cannot be removed.

Treatment.—The habits, hygiene, mode of life, diet, etc., should be most carefully regulated. When the cause or causes are detected they should be promptly removed. The secretions should be kept free at all times. Constipation should be avoided as it serves to embarrass the functional activity of the liver and kidneys. Alcohol should be interdicted. Moderate exercise is of benefit. Potassium iodid and nitroglycerin should be administered in small doses indefinitely. Over-exertion of any kind should be avoided.

ANEURYSM OF THE AORTA.

Varieties.—I. Aneurysm of the arch of the aorta. II. Aneurysm of the thoracic aorta. III. Aneurysm of the abdominal aorta.

The arch of the aorta is divided by Gray into three parts—the ascending, the transverse, and the descending.

The ascending portion is two inches in length, arising from the left ventricle, on a level with the lower border of the left third costal cartilage, behind the left edge of the sternum. It ascends obliquely upward to the right to the upper border of the right second costosternal articulation. The transverse portion commences at the upper border of the right second sternal articulation, and, arching to the left and forward, passes in front of the trachea and esophagus to the left of the third dorsal vertebra. The descending portion extends downward to the left side of the fourth dorsal vertebra.

The thoracic aorta extends from the left lower border of the fourth dorsal vertebra, and ends in front of the body of the twelfth dorsal vertebra, at the aortic opening in the diaphragm.

The abdominal aorta begins at the aortic opening in the diaphragm, descends a little to the left side of the vertebral column, and terminates over the body of the fourth lumbar vertebra, where it divides into the two common iliac arteries.

Definition.—A circumscribed dilatation of some portion of the aorta, the result of disease of the vessel-wall weakening its resistance to the blood-pressure.

Causes.—Conditions that induce arteriosclerosis are the chief causes. Exertion is an exciting cause. Aneurysms occur in early middle life rather than in old age, when the force of the heart has decreased. They are more common in men than in women.

Pathologic Anatomy.—All aneurysms may be divided into two classes, *dissecting* and *circumscribed.* Dissecting aneurysms occur in the aged and result from fatty changes in the internal and middle coats. The intima usually ruptures allowing the blood to dissect its way between the coats of the vessel. Circumscribed aneurysm is most common in middle-aged men and can usually be ascribed to syphilis. It consists of a circumscribed dilatation of the affected vessel. It may be *fusiform, sacculated,* or *cylindric* in shape. A *true aneurysm* is one in which the dilatation is confined to the vessel-walls; a *false aneurysm* is one in which the vessel-wall has ruptured and the extravasated blood has become encapsulated by the adjacent connective-tissue.

ANEURYSM OF THE ARCH.

Symptoms.—This variety of aneurysm is the most common. The onset is usually gradual, with evidences of arteriosclerosis and failing health. Pain, which may be paroxysmal or continuous, is a constant symptom. Dyspnea is also common and may be constant with acute exacerbations, or may be remittent. Occasionally, dysphagia occurs. A slight cough from pressure on the laryngeal nerve, with more or less alterations in the voice, may be present. The pupils are dilated or contracted, or are irregular, in some cases, due to pressure on the sympathetic nerve. There are disorders of the circulation, a gradual loss of flesh, and a careworn expression of the face.

Physical Signs.—*Inspection* is negative until the tumor becomes large when circumscribed bulging and abnormal pulsation may be noticed.

Palpation detects expansile pulsation (Corrigan's sign) which is characteristic. Tenderness may be present over the aneurysm. A thrill may be detected. Diastolic shock due to the recoil of the blood in the aneurysm on the closed aortic valve may also be recognized.

Tracheal tugging is often present and is diagnostic. To obtain it, the patient should be placed in the erect position, with his mouth closed and chin elevated to the greatest extent. The cricoid cartilage should then be grasped between the fingers and thumb and gentle traction upward should be made. The pulsations of the dilated aorta or aneurysm, if any exist, will then be distinctly felt, in most cases transmitted through the trachea to the hand. Aneurysm of the arch also gives rise to alteration in the radial pulse. When the aneurysm is situated at the transverse portion of the arch, the left radial pulse and the left carotid pulse are smaller and weaker than those on the right side.

Percussion yields an abnormal area of dullness with increased resistance.

Auscultation serves to elicit a murmur or bruit over the tumor synchronous with the first sound of the heart. It is louder than the systole, lower in pitch, and of a blowing character. When the aortic valves are intact, the second aortic sound will be markedly accentuated.

Diagnosis.—The presence of a tumor, as shown by the abnormal area of dullness, with expansile pulsation and a bruit in the region occupied by the aortic arch, is diagnostic. The X-ray will serve to define its exact situation and outlines.

ANEURYSM OF THE THORACIC AORTA.

Symptoms.—The most constant symptom is deep-seated thoracic pain, constant or paroxysmal. Dysphagia is a frequent condition. There is seldom dyspnea, and alteration of voice and pupils does not occur. In three cases under my observation, the only symptoms were pain of a deep-seated and boring character in the left shoulder, associated with a feeling of general fatigue. In each, death occurred very suddenly, being preceded by profuse hemorrhage from the mouth. Autopsy revealed a ruptured thoracic aneurysm, the size of a lemon, pressing deeply on the upper left pulmonary lobe.

Physical signs are seldom distinctive and the diagnosis is rarely made during life.

ANEURYSM OF THE ABDOMINAL AORTA.

Symptoms.—The most constant symptom is pain situated in some area corresponding to the aneurysm, or widely diffused over the abdomen. Gastrointestinal symptoms appear and the general health fails. The pressure of the tumor induces retardation of the femoral pulse. Other pressure symptoms depend on the location of the aneurysm. In most cases, it is situated near the celiac axis.

The physical signs reveal abnormal dullness, and the presence of a tumor with expansile pulsation and a bruit to the left of the median line of the abdomen.

Diagnosis.—*Pulsating abdominal aorta* may be distinguished from abdominal aneurysm by its occurrence in paroxysms, in nervous women and effeminate men, and by the absence of a tumor, expansile pulsation, and pressure symptoms.

Abdominal tumors resting on the aorta may transmit its pulsation. The assumption of the knee-chest posture causes the tumor to fall away and the pulsation is lost.

Prognosis of Aortic Aneurysm.—Unfavorable. The duration of life after the development of the aneurysm is from one to four years. The termination may be sudden from rupture and hemorrhage or gradual from exhaustion.

Treatment.—The object of the treatment is to promote coagulation of the blood within the sac and to bring about contraction of the tumor, at the same time being careful to avoid violent rupture.

The so-called Tufnell's method is the most successful for these purposes, its aim being to diminish the force and rapidity of the circulation, and, if possible, to increase the fibrinous deposit. Its essential element is absolute rest of mind and body, and a restricted diet; the patient is kept absolutely in bed day and night for at least three months, and placed on the following diet: Breakfast—two ounces of bread with butter and two ounces of milk; dinner—two or three ounces of bread, same amount of meat, and two to four ounces of milk or claret wine; supper—two ounces of bread with butter and two ounces of milk. At the same time potassium iodid is administered in increasing doses to the physiologic limit.

Galvano-puncture is said to do good in some cases; two needles inserted into the aneurysm are connected with the poles of a galvanic battery, and a weak current is passed through the tumor. Various surgical procedures have been employed, from time to time, but the success following them is doubtful.

The severe pain indicates the use of morphin and the local appilcation of an ice-bag. Cyanosis and dyspnea will be relieved to some extent by venesection.

DISEASES OF THE NERVOUS SYSTEM.

GENERAL SYMPTOMATOLOGY.

Motor Phenomena: The motor disturbances incident to nervous diseases may be manifested as paralysis or loss of motion, or as excessive motor discharges including convulsions, tremors, and choreiform movements.

Paralysis involving a lateral half of the body is termed ***hemiplegia;*** when involving the body from the waist down, ***paraplegia,*** and when involving a single member, ***monoplegia.***

Paralysis may be irregularly distributed and in such cases may be due to localized disease of the muscles or nerves of the affected region, or to syringomyelia, disseminated lesions in the motor area of the brain, lesions of the basal ganglia, and poliomyletis (acute and chronic).

Hemiplegia usually results from hemorrhage at the base of the brain injuring the internal capsule, corpus striatum, or optic thalamus. The paralysis occurs on the opposite side of the body. As other causes of unilateral paralysis may be mentioned lesions of the motor cortex, crus cerebri, or pons, a unilateral lesion high up in the spinal cord, and hysteria.

Paraplegia may be due to multiple neuritis, caisson disease, or hysteria but in most cases is the result of injury or disease of the spinal cord such as occurs in fracture or caries of the vertebræ, morbid growths, aneurysm, hemorrhage, acute myelitis, chronic myelitis, Landry's disease, and lateral sclerosis. Injury to the brain during delivery may induce spastic paraplegia.

Monoplegia may result from disease or injury of a peripheral nerve, a focal lesion in the cortex, or from hysteria.

Convulsions may be defined as general involuntary paroxysms of muscular contraction. They may consist of continuous contractions, *tonic,* or intermittent contractions, *clonic.* They may be general or local. They are usually considered as of three varieties *epileptiform, tetanic,* and *hysteroidal.*

Epileptiform convulsions may be observed in epilepsy, organic brain disease, cerebral anemia, uremia and other toxemias such as eclampsia, infectious fevers, etc., and reflex conditions, especially those referable to the digestive tract. Unconsciousness is usually present and the contractions are mostly clonic.

Tetanic convulsions occur in tetanus, tetany, spinal meningitis, and strychnin poisoning. Consciousness is retained.

Hysteroidal convulsions follow no fixed rule. Consciousness is never entirely lost. Other hysteric manifestations are present and the patient never inflicts injury upon herself.

Tremors are involuntary vibratory movements and are produced by alternate contraction and relaxation of antagonistic muscles. They are observed most often in the arms, head, face, tongue, and hands. They may be *coarse* or *fine.* Tremors occur in chronic alcoholism, delirium tremens, paralysis agitans, and in poisoning by lead, mercury, arsenic, chloral, and opium. Neurasthenia, debility from various causes, senility, hysteria, disseminated sclerosis, and paresis are accompanied by tremors. In *disseminated sclerosis,* the tremor is irregular, jerky, and increased by voluntary efforts to restrain it. The tremor is absent during rest but is brought about by movement. In *paralysis agitans,* it is regular and rhythmic, occurring both during rest and movement. The tremor of *senility* is exceedingly fine and begins in the hands often extending to the face. It occurs at first only during motion, disappearing during rest. When age is far advanced it may occur during both rest and movement.

Choreiform movements are coarse, incoördinated involuntary movements of a jerky and irregular character usually separated by short intervals. They may simulate, to some extent, purposeful movements. Among the causes may be mentioned idiopathic chorea, Huntingdon's chorea, posthemiplegic chorea, organic brain disease, habit, hysteria, reflex irritation, etc.

Athetoid movements are slow, more or less rhythmic twisting movements of the fingers and toes. They are observed in cerebral palsies of children, after hemiplegia in adults, and poliencephalitis.

The gait may also be taken as an index of the character of the nervous condition present. The *ataxic gait* is especially characteristic of locomotor ataxia. In it, the patient raises the foot very high, throws it outward and forward, and allows it to fall suddenly to the ground in an awkward manner. The *spastic gait* observed in spastic paraplegia is characterized by stiff movements of the lower extremities. The knees are somewhat flexed and approach each other, and the toe drags on the ground with each step. The *festinating gait,* or the gait of paralysis agitans in the later stages is distinguished by the following features: As the patient walks, the body bends forward and the steps follow each other in rapid succession until the patient falls or supports himself by means of some nearby object. After a very short interval in which equilibrium is obtained, the patient repeats the cycle. The *steppage gait* is that in which the foot is highly elevated and the toe turned up in taking a step. In bringing the foot down the heel is first placed on the ground. This gait occurs in multiple neuritis. *Titubation* is the term applied to that gait in which there is considerable swaggering and swaying, particularly that gait occurring in disease of the cerebellum.

The reflexes are motor phenomena to which the attention should always be directed in considering diseases of the nervous system. They are of two kinds: *cutaneous reflexes* and *tendon reflexes.*

The *cutaneous reflexes* are superficial reflexes and consist of muscular contractions produced by irritation of the sensory nerves in the skin. The contractions induced by tickling the soles of the feet may be mentioned as examples. Various names are applied to these reflexes according to the situations in which they occur. Cutaneous reflexes may be delayed in certain nervous diseases and in others the response to irritation may be prompt and extend over the entire body. They are absent in shock, diseases of the brain and spinal cord involving the reflex centers, and diseases of the peripheral nerves. They are increased in affections in which there is increased irritability of the cutaneous nerves as in tetanus, strychnin poisoning, general neuroses, etc.

The *tendon reflexes* are the muscle contractions produced by gently tapping the tendons while the corresponding muscles are placed slightly upon the stretch. The same effect, but of less intensity may be produced by striking the adjacent fascia and periosteum.

The *knee-jerk* or patellar reflex is produced by striking the tendon of the quadriceps extensor muscle between the patella and its insertion while the patient crosses the leg loosely over the opposite knee or allows it to hang, relaxed, over the forearm of the examiner. Simultaneous muscular effort on the part of the patient will serve to increase the reflex. The knee-jerk is increased in lateral sclerosis, diseminated sclerosis, incomplete lesions of the cord above the lumbar segment, irritability of the spinal cord as occurs in spinal meningitis, strychnin poisoning, hysteria, etc., and in some cases of organic cerebral disease. It is diminished or absent in locomotor ataxia, neuritis, pseudomuscular hypertrophy, poliomyelitis, myelitis, and in poisoning by spinal depressant drugs. Pronounced physical exhaustion also serves to lessen the tendon reflexes.

Ankle-clonus is the term applied to the vibratory movements of the foot produced by forcible dorsal flexion of the foot. It is seldom if ever obtained during health, being observed most often in lateral sclerosis and hysteria.

The *Babinski reflex* is the extension of the great toe which follows tickling the sole of the foot. Normally flexion follows such a procedure. The reflex occurs most often in hemiplegia, diplegia, and diseases of the motor tract of the cord. Disease of Lateral Col.

Other reflexes occur in connection with the arm, *arm-jerk,* and the jaw, *jaw-jerk,* and are obtained by striking their respective muscles while in a state of partial extension. The contraction of the pupil on exposure to light, the closure of the eyelids on irritation of the cornea or conjunctiva, sneezing following irritation of the nares, and other similar reflexes are entitled to mention in this connection.

Paradoxic Contraction consists of a tetanic contraction of the tibialis anticus produced by forcibly flexing the foot on the leg. The foot remains flexed for several minutes after which it slowly relaxes. It was first described by Westphal, and may be observed in tabes dorsalis, hysteria, paralysis agitans, and multiple sclerosis. The phenomenon may occasionally be produced in the flexors of the leg and forearms.

Vasomotor Disturbances.—Paralysis of the vasomotor system occurs as a symptom of hysteria, neurasthenia, and other functional neuroses and follows injuries of the sympathetic nerve. It is manifested by abnormal redness of the skin with a sensation of heat and a rise in the dermal temperature. Vasomotor spasm is indicated by pallor and coolness of the skin with formication and stiffness. It is observed with functional disturbances of the sympathetic system and may be followed by trophic disturbances such as occur in scleroderma and symmetric gangrene.

Sensory Phenomena.—Sensibility may be increased, ***hyperesthesia***, decreased or absent, ***anesthesia***, or perverted, ***paresthesia***. In hyperesthesia, the increase is so great that even the most slight irritation may produce pain.

It may be due to inflammation of the nerves as in sciatica and multiple neuritis when it is associated with tenderness on pressure. It is also present in inflammation of the meninges, neurasthenia and hysteria.

Anesthesia of the skin may be complete or partial and results from any disturbance in the conducting path from the body surface to the centers of sensation. As causes, may be mentioned neuritis, traumatism of the nerve-trunks, organic disease of the sensory tract in the spinal cord or brain, hysteria, reflex irritation, drugs such as morphin, cocain, and other local anesthetics, and caustics such as the mineral acids, alkalies, carbolic acid, etc.

Hemianesthesia is the term applied to loss of sensation on a lateral half of the body. It is usually associated with hemiplegia on the same side except when due to a unilateral lesion of the cord in its upper segment, when the hemiplegia is on the opposite side. The condition is due in most cases to hemorrhages, tumors, or local softening of the posterior limb of the internal capsule, the crus or peduncle, the pons, the medulla, or the occipital cortex. Hysteria is responsible for a number of cases of hemianesthesia. These cases are paroxysmal in character and unassociated with loss of motion.

Monanesthesia is used to denote loss of sensation in a single member. Inflammation, injury, or other lesion of the corresponding sensory nerve is the most common cause but it may also arise from focal lesions in the occipital cortex and hysteria.

Paranesthesia signifies absence of sensibility of the body and extremities below the waist. Organic disease of the spinal cord and neuritis of the large sensory nerve trunks of the lower extremities are the most common causes. Hysteria and reflex irritation may, however, greatly influence its production.

Thermoanesthesia is a variety of diminished sensibility of the skin in which it is impossible to appreciate heat or cold by tactile impression. In health, it is possible to recognize differences of 2° F. on the back, and differences of ½ to 1° F. on the fingers and face at temperatures from 80° to 100° F. In disease, the temperature sense may be lost while other forms of irritation are appreciated. It may be observed in syringomyelia and hysteria.

Analgesia is that condition in which there is insensibility to pain as produced by pinching a fold of skin, pricking with a needle, or by electric currents. Tactile insensibility does not necessarily imply analgesia. Loss of sensibility to pain is a prominent feature of syringomyelia, but may also be observed in peripheral and central nervous disease and in hysteria.

Delayed conduction of sensory impulses is frequently observed in anesthesia from various causes. In locomotor ataxia, it is particularly common.

After-sensations are the painful sensations which succeed momentary impressions such as follow the prick of a pin. Sometimes an isolated prick of a pin is not appreciated requiring repetition of the procedure several times, after which, with a varying interval, the painful sensations present themselves. This phenomenon is especially frequent in locomotor ataxia and other diseases of the cord and of the nerves.

Paresthesia is applied to abnormal sensations in the skin such as numbness, tingling, itching, pricking, formications, etc. It is observed in numerous conditions of the nervous system especially neurasthenia, hysteria, spinal sclerosis, and disturbances of the peripheral nerves. The girdle sensations which occur in locomotor ataxia and other forms of spinal sclerosis belong to this class of sensory phenomena.

Neuralgia is the term applied to paroxysmal pain occurring along the line of the nerve-trunks. Pressure usually serves to relieve it. Points of tenderness may, however, be elicited where the affected nerve emerges from a bony canal or from beneath muscular coverings. The *lightning-pains* observed in locomotor ataxia are sharp, lancinating, neuralgic pains occurring usually in the extremities. *Causalgia* is an intense burning neuralgia and is encountered most frequently in the condition known as "glossy skin."

Nutritive Disturbances.—*Atrophy of the muscles* may result from acute or chronic anterior poliomyelitis, inflammation or injury of the nerves, idiopathic muscular disease, or disease such as follows cerebral palsies and joint affections.

Degeneration of the muscles may be determined by their reaction to the galvanic electric current. In the normal muscle the cathodal closing contraction is greater than the anodal closing contraction and cathodal opening contraction is less than the anodal opening contraction. In the early stages of degeneration, the anodal and cathodal contractions are equal, both on opening and on closing the current. When the degeneration is advanced, cathodal closing contraction is less than anodal closing contraction, and cathodal opening contraction is greater than anodal closing contraction. This, it will be noticed, is a reversal of the reaction of normal muscle. These degenerative reactions indicate suspension of trophic influences and are symptomatic of acute and chronic anterior poliomyelitis, acute central myelitis, and inflammation, traumatism, or other disturbance of the nerves which arrests their functions.

X Normal = KCC > ACC
+ AOC > KOC

anced - KCC < ACC

Arthropathies may occur in certain organic diseases of the nervous system such as syringomyelia and locomotor ataxia, and consist of swelling, effusion, and degenerative changes in the joints.

Ulceration may result in the course of certain diseases of the nervous system from coincident disturbance of nutrition. When ulceration occurs on parts subjected to pressure, within a few days, the term *acute decubitus* may be employed to express the condition; when ulcerative lesions appear after a long lapse of time in chronic nervous diseases, the term *chronic decubitus* is applied. *Perforating ulcer of the foot* such as occurs in locomotor ataxia may also be mentioned in this connection. Somewhat allied to ulceration due to nutritive disturbances is *spontaneous gangrene* (*Raynaud's Disease*) which involves the fingers, toes, ears, nose, etc., in the absence of any local causes.

Trophic disturbances involving the skin and its appendages include scleroderma, chloasma, vitiligo, atrophia unguis, plica, trichorrhexis nodosa, etc.

Disturbances of Consciousness.—The principal alterations to which consciousness is subject in nervous diseases are coma, trance, somnambulism, ecstasy, and catalepsy.

Coma is an abnormally deep and prolonged sleep in which the cerebral functions are in abeyance, characterized by stertorous breathing, relaxation of the sphincters, lividity of the face, loss of parallelism of the optic axes, and an inability to respond to external stimuli. It may be gradual or sudden in its onset; complete or partial, transient or permanent. It may be due to organic brain disease, traumatism, cerebral anemia, epilepsy, sunstroke, hysteria, various convulsive states, and various toxic agents in the blood, introduced either from without or produced within the body.

Trance is an hysteric manifestation characterized by a prolonged abnormal sleep from which the patient cannot be aroused and in which the vital functions are reduced to a minimum.

Somnambulism is a condition of half-sleep in which the senses are but partially suspended and the patient is able to perform various feats automatically. Ordinary sleep-walking may occur in health but the more pronounced varieties of this condition are observed in hysteria and in hypnotized subjects.

Ecstasy is a peculiar state of the mind in which a delusion so governs the mental functions that the entire nervous system is held in a condition of subjection or apparent insensibility. It is usually an hysteric manifestation.

Catalepsy is characterized by loss of will and by muscular rigidity. It occurs in paroxysms with loss of consciousness, the limbs remaining for long periods in any position in which they are placed. It occurs in hysteria, various psychoses, hypnotic states, and organic brain disease.

Disturbances of the Special Senses.—*The eye* frequently shows manifestations of general nervous diseases that are to some extent characteristic. *Myosis* or contraction of the pupil occurs in paresis, locomotor ataxia, meningitis, brain tumor, disseminated sclerosis, uremia, and other similar conditions while *mydriasis* or dilatation of the pupil may be observed in optic atrophy, paralysis of the third nerve, epileptic and hysteric attacks, paresis, locomotor ataxia, etc. *Unequal pupils* may be seen in health and in local ocular disease in addition to paretic dementia, locomotor ataxia, and affections interfering with the nerve-supply of the iris.

The *Argyll Robertson pupil* is that which fails to respond to light but accommodates for distance. It is symptomatic of paresis and locomotor ataxia.

X *Conjugate deviation of the eyes* consists in outward rotation of the eyes, such as occurs in apoplexy and cerebral convulsions of organic origin.

Nystagmus or tremor of the eyeball, when unassociated with local ocular disease may be taken as an indication of disseminated sclerosis, Friedreich's ataxia, or affections of the basal ganglia.

Optic neuritis, papillitis, or choked disk, occurs in the course of tumors and cerebral meningitis. It may also be produced by Bright's disease, syphilis, anemia, and various toxic conditions.

Primary optic atrophy is especially significant of locomotor ataxia and paresis. Secondary optic atrophy is usually due to inflammation, injury, tumor, etc., of the optic nerve.

× Both eyes turned strongly & persistently to one side, either right or left – conjugate deviation
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The ear is also affected in the discharge of its function in certain nervous diseases but to a less extent. Deafness may be due to affections of the auditory nerve in some part of its course but it usually secondary to some local condition. Exaggeration of the hearing occurs in cerebral hyperemia and hysteria. *Tinnitus aurium*, or ringing in the ears, arises from local ear disease, cerebral hyperemia and anemia, and Menière's disease, and after the use of certain drugs, such as quinin and its derivatives and the salicylates in excess.

Mental Phenomena.—An *hallucination* is a state of the mind in which the patient believes he perceives external objects that do not exist, or in other words is a condition of false perception occurring independent of external impressions.

A *delusion* is a faulty belief concerning a subject capable of physical demonstration, out of which the patient cannot be reasoned by adequate methods for the time being (H. C. Wood).

An *illusion* is a perverted impression based upon an actual perception.

Delirium is a condition of mental aberration characterized by an apparent exhaltation of all the processes of the mind manifested by mental irritation and confusion, transitory delusions, and fleeting hallucinations, and by disordered, senseless speech, and by motor unrest. It may be a part of mania, hysteria, or acute mania, or it may be secondary to some toxic condition such as accompanies uremia, infectious fevers, alcoholism, etc.

Diseases of the Nervous System may be conveniently studied under the following headings:

I. Diseases of the Cerebral Membranes. II. Diseases of the Cerebrum. III. Diseases of the Spinal Cord. IV. Diseases of the Nerves. V. General Nervous Diseases. VI. Mental Diseases.

DISEASES OF THE CEREBRAL MEMBRANES.

The brain is invested with two membranes—the dura mater and the pia mater, or pia-arachnoid.

The *dura* lines the interior of the skull, and, in addition, supports and protects the brain. The *falx* is an extended process of the dura which extends into the longitudinal fissure; the *tentorium* is a process of the dura separating the cerebrum and the cerebellum; the *falcula* is a process of the dura extending between the two hemispheres of the cerebellum.

The blood-supply for the dura is from the anterior, middle, and posterior meningeal arteries. The middle meningeal or medidural artery, a branch of the internal maxillary, is the largest of the three, and is the vessel usually involved in meningeal hemorrhage.

The nerve-supply (a mooted question) is undoubtedly received from the fifth or trigeminus pair of cranial nerves, irritation of which nerve-supply may produce *hyperesthesia, pain, reflex motor,* and *vaso-motor* disturbances (Duret). The *pia* (which includes the *arachnoid,* after the suggestion of Tuke, and which Mills calls the *arachnopia,* or *pia-arachnoid*) is composed of two layers—the *visceral* layer and the *parietal* layer. This membrane is a vascular network held by connective-tissue. The *visceral* layer of the pia (formerly known as the pia alone) closely invests the brain everywhere, dipping into the fissures and into the ventricles. The *parietal* layer (formerly known as the arachnoid) closely covers the dura in all its parts.

The *pia-arachnoid* is the nutritive covering of the brain, supplying a considerable section with blood. The vessels of the pia lie on the surface and are encased in perivascular sheaths composed of the denser portions of the membrane. These perivascular spaces are the lymph-canals accompanying the blood-vessels into the brain-substance and communicating with the subarachnoid spaces or cisterns.

The nerve supply of the *pia-arachnoid* is still in dispute, the membrane being generally considered without sensation. This is probably an error.

The *Pacchionian* granulations are always present in abundance "on the outside of the dura, on its inner surface, on the arachnopia, and within the superior longitudinal sinus and the parasinoidal spaces, or lacs sanguinis. They often indent the calvarium, and in rare instances they penetrate it. It is generally conceded that they are enlargements of the normal villi or tuft-like elevations of the parietal layer of the pia (arachnoid). Repeated attacks of meningeal hyperemia probably assist in their development" (Mills).

PACHYMENINGITIS.

Synonyms.—Meningitis; hematoma of the dura mater.

Definition.—Inflammation of the *dura mater;* when the external layer is primarily involved, it is termed *pachymeningitis externa;* when the internal layer is primarily involved, it is termed *pachymeningitis interna.*

Causes.—*Pachymeningitis externa* is a surgical malady, resulting from fractures, penetrating wounds, and other injuries of the skull.

Pachymeningitis interna may be due to blows upon the head without injury to the skull, chronic alcoholism, scurvy, Bright's disease, tuberculosis, and syphilis. Chronic internal otitis and suppurative inflammation of the orbit may cause it, as may also inflammation in the venous sinuses the result of a thrombus undergoing suppurative changes. Erysipelas, sun-stroke, and gout are recorded causes.

Pathologic Anatomy.—*Pachymeningitis interna* begins with hyperemia of the membrane, followed by an exudation which develops into a membranous new formation, containing a great number of vessels of considerable size, but having very thin walls. Hemorrhages from these new vessels are of frequent occurrence, which increase the size and thickness of the neo-membrane.

The usual position of the neo-membrane or new formation is on the upper surface of the hemispheres, extending downward toward the occipital lobe. The changes in the adjacent portion of the brain are dependent on the size and thickness of the neo-membrane. Bartholow observed a case in which the "cyst" was half an inch in thickness at its thickest part, and it depressed the hemisphere correspondingly, the convolutions being flattened, the sulci almost obliterated, and the ventricle lessened one-half in size.

In *pachymeningitis syphilitica,* the pathologic lesion is in the form of gummatous tumors or masses which may degenerate and become either cheesy masses or be converted into a purulent-looking fluid.

In *old age* the dura mater becomes thick, cartilaginous, and of a dull white color. The sheaths of the arteries are also thickened.

Symptoms.—These are very obscure and are principally those of cerebral compression. Persistent headache, vertigo, photophobia, insomnia, and gradual impairment of intellect and locomotion followed by delirium, convulsions, and coma, or by apoplectic attacks and paralysis, occurring in the aged or those in whom any of the already-mentioned causes exist, should lead the examiner to suspect inflammation of the dura mater. Epileptic attacks sometimes occur in this condition. Circumscribed painful edema behind the ear and less fullness of the corresponding side, the *phlegmasia alba dolens en miniature* of Griesinger, are indicative of thrombosis of the transverse sinus, a condition nearly always accompanied by pachymeningitis.

Diagnosis.—The diagnosis is always difficult and frequently impossible on account of the obscurity of the symptoms. Many cases are recognized only at autopsy.

Prognosis.—The outlook is unfavorable in all forms. In traumatic cases, surgical intervention offers some hope of cure.

Treatment.—Pachymeningitis externa is to be treated surgically. Trephining is indicated in some cases. It is claimed that benefit has followed a thorough course of potassium iodid. In the great majority of cases, however, all that can be done is to treat symptoms as they arise.

ACUTE LEPTOMENINGITIS.

Synonyms.—Acute meningitis; cerebral fever; arachnitis.

Definition.—An acute exudative inflammation of the cerebral pia mater and arachnoid membranes (pia arachnoid, or arachnopia), usually limited to the convexity of the cerebrum; characterized by fever, vomiting, headache, and delirium, followed by symptoms of general collapse.

Causes.—It may occur during the course of the infectious fevers, especially erysipelas, typhoid fever, influenza, pneumonia, and diphtheria, or it may follow middle-ear disease and injury or disease of the cranial bones It may be secondary to some tuberculous focus elsewhere in the body or to disease of other serous membranes. Among other causes may be mentioned cerebral overwork, prolonged wakefulness, acute alcoholism, exposure to the sun, and syphilis. In rare instances the disease may occur as an independent affection. The condition occurs most frequently in children and young adults, affecting males more often than females.

The exciting cause is a microörganism. In the primary variety, the diplococcus intracellularis is the exciting cause; in the secondary forms, the microörganism with which the underlying cause is associated, especially the pneumococcus, streptococcus, typhoid bacillus, tubercle bacillus, and the diphtheria bacillus.

Pathologic Anatomy.—The inflammatory changes may be limited to the convexity or to the base of the brain but more frequently both portions are involved. The earliest change is hyperemia which is soon followed by turbidity and opacity of the affected membrane. As the process advances a sero-purulent, purulent, or fibrinous exudate is formed which distends the subarachnoid space and may fill the ventricles thereby compressing and flattening the convolutions. The condition may extend to the brain substance. In fourteen out of twenty-four cases at the Philadelphia Hospital, meningo-encephalitis was revealed at autopsy. When due to some general infection the inflammation is more or less diffused over the entire brain, but when secondary to some local infection as middle-ear disease and tuberculosis it is basilar and to some extent circumscribed. The tubercular form is characterized by the formation of small tubercles and a yellowish gelatinous material. When the ventricular effusion is very great it constitutes *acute hydrocephalus.*

Symptoms.—The onset may be sudden but is usually gradual accompanied by such prodromes as persistent headache, vertigo, irritability of temper, vomiting without nausea, feverishness, coated tongue, and constipation. These symptoms may continue from a few hours to two or three days.

The *stage of invasion* is manifested by chill, high fever, 103°-104° F., rapid pulse, 100-120, flushed face, congested eyes, intense continuous headache, ringing in the ears, photophobia, vertigo, nausea, aggravated vomiting, delirium, and general cutaneous hyperesthesia.

The *stage of excitation* is characterized by increased cutaneous sensibility, increased sensitiveness to light and sound, furious delirium often resembling mania, continual jerking of the limbs, oscillation of the eyeballs (mystagmus), twitching of the facial and other muscles, retraction of the head, arching backward of the body, and sometimes convulsions. The pulse is slow and irregular, and the fever is high. Headache continues and may be subject to exacerbations during which the patient cries out in a peculiar manner (*the hydrocephalic cry*). Coated tongue, constipation and retraction of the abdomen are present. The finger drawn across the abdomen leaves a red line, the *tache cérébrale.* The duration of this stage is from one day to one or two weeks.

The *stage of depression* or collapse appears as the exudate accumulates in sufficient quantities to induce marked pressure and is manifested largely by paralytic phenomena. The patient gradually becomes more quiet, the muscular agitation and delirium subsiding. Somnolence develops and passes into coma; at times there is temporary consciousness soon followed by coma. The pulse is slow and irregular and the fever is lessened. Various palsies such as strabismus, ptosis, paralysis of pupillary reaction, and relaxation of the sphincters are present. Cutaneous anesthesia, blindness, deafness, and Cheyne-Stokes breathing eventually supervene. Death ultimately follows, being ushered in with convulsions or coma with cyanosis.

Diagnosis.—The characteristic symptoms of acute leptomeningitis are rapidly developed headache, vomiting unassociated with nausea or gastric trouble, fever, and delirium.

Cerebrospinal fever may be distinguished by the marked spinal symptoms, the eruption, the presence of the diplococcus intracellularis in the fluid withdrawn by lumbar puncture, and its occurrence in epidemics.

Tubercular meningitis is attended by symptoms referable to disease at the base of the brain and by the symptoms of tuberculosis elsewhere in the body. Its onset is slow.

Cerebral complications in typhoid fever, typhus fever, rheumatism, pneumonia, etc., may be confused with acute leptomeningitis but a careful study of the history, symptoms, etc., will serve to make a distinction.

Uremia differs from acute leptomeningitis in that the face is turgid and edematous; the eyelids are puffy; albuminuria is constant; an irregular temperature is present; and convulsions are common, while in leptomeningitis the face is pale, edema is absent; albuminuria seldom occurs; and the attack begins with chills followed by fever.

Delirium tremens is characterized by busy delirium, the patient imagining that he is surrounded with persons and animals and is wild in his gestures and utterances; the temperature is normal or subnormal and the skin is wet and clammy. In leptomeningitis the delirium is mild but incoherent, the surface is hot and dry and there is severe vomiting and headache.

Prognosis.—The outlook is very unfavorable. If recognized early and treated, a fair number of recoveries occur, but it usually leaves the patient subject to attacks of epilepsy or with a persistent headache, and more or less mental impairment. Blindness and chronic internal hydrocephalus are rare results. The duration is from a few days to two or more weeks.

Treatment.—Keeping in view the course and general prognosis of leptomeningitis, it is questionable if any very active medication will abate the disease during any stage. Absolute rest in a quiet, well-ventilated room with the head elevated will, however, serve to lessen the severity of the symptoms. The diet should be liquid in character; all the secretions should receive attention; and an ice-bag should be applied to the head to relieve the intense headache. In vigorous or sthenic cases, with high febrile reaction and exaggeration of the early symptoms, venesection or leeches behind the ears, to the temples, or in the nuchal region may be employed, followed by the application of cold and the internal administration of fluid extract of ergot in large doses every two hours. The cerebral circulation may be markedly influenced by compression of the carotids. Vomiting may be satisfactorily relieved in nearly all cases by the use of chloral, gr. iii-v (0.2-0.3 Gm.), diluted, every half hour, by the mouth until relieved, or in doses of from gr. x-xv (0.6-1 Gm.), by the rectum. The restlessness, convulsions, delirium, etc., require the use of morphin, bromids, chloral, phenacetin, and similar drugs. Temperature may be reduced by hydrotherapy. The course of the disease may be greatly influenced by lumbar puncture. The various preparations of mercury are often of great value, particularly in chronic cases. In the late stages, tonics and stimulants should be freely given combined with the use of potassium iodid and iodid of iron, and the application of flying blisters.

TUBERCULAR MENINGITIS.

Synonyms.—Basilar meningitis; acute hydrocephalus.

Definition.—An inflammation of the leptomeninges (soft membranes), particularly the basal pia mater, attended with or due to the deposit of gray miliary tubercle, characterized by gradual decline of the bodily and mental powers in addition to symptoms referable to meningeal inflammation.

Causes.—It usually occurs as a secondary affection; commonly a sequel to tubercular disease of some other organ. It is observed most frequently in children between two and six years of age, although numerous cases are reported as having occurred between the ages of twenty and thirty years. The influence of the scrofulous diathesis, so-called, in the production of this affection is very great. The "gelatinous children of albuminous parents," as the phrase goes, possess a special susceptibility for tubercular meningitis.

Pathologic Anatomy.—The deposition of tubercle usually occurs at the base of the brain. Depositions of grayish-white granules, of a translucent, somewhat gelatinous appearance—miliary tubercle, are distributed along the vessels of the pia mater, resulting in inflammation and the exudation of lymph, with the consequent thickening and opacity of the membranes. The cerebral tissue is not usually involved, although on section the lines indicative of blood-vessels are very much increased in number. The ventricles are distended by a clear, or milky, or even bloody serum. The presence of the tubercles alone may give rise to no symptoms until the exudative products of the resultant inflammation develop.

Tubercular deposits occur also in the lungs, intestines, and, at times, in other organs.

Symptoms.—The onset may be sudden or gradual. Convulsions may usher in the attack. Prodromal symptoms are usually present. The child becomes irritable and there are present anorexia, loss of flesh, swelling of the abdomen, constipation alternating with diarrhea, irregular periods of fever with grinding of the teeth, and sleeplessness. Headache occurs as is shown by the child, even when at play, stopping and resting its head on its hand or on the floor. The duration of this stage is from one week to one or two months.

The *stage of excitation* begins suddenly with obstinate vomiting, severe headache, convulsions, fever, 102°-103° F., in the evening falling to 99° F., in the morning, and a soft and compressible, irregular pulse. On drawing the finger nail lightly over the surface of the body a red line results, "the cerebral stain," of Trousseau. The special and general senses become exalted, resulting in photophobia, tinnitus aurium, intolerance to sound, and cutaneous hyperesthesia. The muscles are subject to spasmodic contraction and rigidity, at times giving rise to opisthotonos. This period of the disease lasts from two weeks to a month.

The *stage of depression* follows the preceding and is the result of the pressure of the exudation on the brain. The pulse is slow and compressible and its rhythm irregular. The temperature becomes less. A tendency to somnolence alternating with quiet delirium soon becomes manifest. There are also continual movements of the fingers, as in picking up objects, mental stupor, periodic convulsions, strabismus, and oscillation of the eyeballs. Intervals of wakefulness occur during which the headache becomes excruciating, causing the peculiar shrill cry or shriek, "the hydrocephalic cry." These are associated with contraction of the facial muscles as if intense suffering were experienced. Collapse finally occurs with Cheyne-Stokes breathing and deepening coma which eventually terminates in death with or without convulsions. The duration of this stage is from one or two days to a week.

Diagnosis.—*Acute leptomeningitis* and tubercular meningitis have closely analogous symptoms during the stage of excitation, but the history and clinical course of the two maladies determine the diagnosis.

Prognosis.—Unfavorable. The usual duration is three or four weeks after fully developed prodromes. If ushered in by convulsions, the duration is shorter.

Treatment.—There are no means of retarding the disease. The measures recommended under Acute Leptomeningitis may be of service in rendering the patient more comfortable. Cod-liver oil, syrup of the iodid of iron, syrup of hydriodic acid, and quinin should also be administered.

DISEASES OF THE CEREBRUM.

To understand the symptoms in diseases of the nervous system, a clear and precise knowledge of the anatomy and physiology is necessary. Presuming this knowledge, only a very few of the most elementary facts will be mentioned before discussing diseases of the brain and cord.

The *nerve-cell* is the real foundation of the nervous system. It receives its nourishment from the arterioles and the lymphatics, and is drained by the venules, as are other tissues, and is supported by the connective-tissue. Each nerve-cell has two processes, the *axis cylinder process* and the *protoplasmic process;* the three—the cell and the two processes—are known as the *neuron,* the entire nervous system being made up of neurons. The axis-cylinder processes conduct the nerve impressions or current from the cells, and constitute a continuation of the axis-cylinder of the nerve. The protoplasmic process conducts the nervous current or impressions into the cell, and it is through these processes and their collaterals that the cell is brought into communication with all portions of the body. The nerve-cells—"the very inner citadel of nervous life"—are mainly set in the gray matter of the brain and the spinal cord, and the axis-cylinder processes and the protoplasmic processes run in bundles or collections in the white matter of the brain and spinal cord. The gray matter of the brain and spinal cord, or the nerve-cells, is found chiefly in the cortex of the cerebrum and the basal ganglia, in the cortex of the cerebellum, in the horns of the spinal cord, and, in the nuclei of the medulla oblongata, and all these masses of gray matter or cells are connected by nerves, or white matter, each protected by connective-tissue. The cells endow the nerves with their particular functions. A knowledge of the physiology of the nervous system is essential in order to understand the alterations in the functions of the different masses of gray matter, or cells, and of the nerves, or white matter.

A knowledge of the blood-supply of the brain is of great practical importance, and particularly for the understanding of the symptoms and pathology of apoplexy and cerebral emboli.

The external carotids on each side, supply blood to the scalp the skull, and the dura mater.

The internal carotid artery on each side, and the veretebral arteries supply the brain, pia mater, and the eyes.

The internal carotid arteries divide into the anterior cerebral and the middle cerebral arteries.

The vertebral arteries on each side give off the inferior cerebellar arteries, and then join and form the basilar artery, which divides, forming the two posterior cerebral arteries, which, in turn, give off a posterior communicating artery. It is the union of these cerebral arteries by the anterior and posterior communicating arteries that forms the circle of Willis. From various portions of the circle of Willis and the beginnings of the anterior, middle, and posterior cerebral arteries are given off six groups of vessels, which furnish the blood-supply to the basal ganglia and the adjacent white matter, from which they derive their name, "the central arteries of the brain." The "central arteries" given off by the middle cerebral or Sylvian artery are of the most importance to the clinician. They are known as the lenticular-optic and the lenticular-striate arteries, and are usually involved in cerebral hemorrhage.

Without a knowledge of the known centers of "localization" it is impossible to interpret the symptoms of diseases of the nervous system.

The motor area is made up of the ascending frontal and ascending parietal convolutions, the posterior part of the three frontal convolutions, the upper part of the parietal lobe adjoining the ascending parietal convolution, and the paracentral lobe. This region may be further "localized" for separate groups of muscles.

The center for muscular sense is believed to be located largely in the parietal lobe.

The sensory areas are located in the cortex but their exact situation is not absolutely proved. Many observers believe them to be localized in the motor area while others contend that the sensory center is in the hippocampal convolution, and still others in the gyrus fornicatus.

The auditory center is located in the first temporal gyrus.

The visual center is in the occipital lobe and its cortical area in the cuneus and adjacent convolutions.

The speech center is located in the third left frontal and first temporal convolutions in right-handed individuals and in similar convolutions on the right side in left-handed persons. The various phenomena resulting from injury or disease of this area are termed collectively *aphasia*. Tyson describes it as a loss of power to comprehend words correctly and to use them properly. It may be subdivided into mind-blindness, *apraxia*, word-blindness, *alexia*, loss of memory for words, *amnesia*, word-deafness, etc.

The "mind" center has long been considered as located in the frontal lobe, anterior to the motor area and the third frontal convolution, but of late the view is growing that for complete integrity of the mind the entire cortex must be intact, although lesions of the portions named, produce mental symptoms only, while lesions of other portions of the cortex cause other disorders in which mental changes are more or less prominently observed.

The many symptoms resulting from diseases of the brain can be placed in four groups:

1. General symptoms of brain irritation. 2. General symptoms of brain-pressure. 3. Symptoms of focal irritation or destruction. 4. Symptoms due directly to the pathologic process.

Symptoms of brain irritation, or hyperemia, are: headache, vertigo, vomiting, photophobia, mental irritability, insomnia, fullness or pressure over the brain, with scalp tenderness and noises in the ears. Rarely convulsive symptoms and delirium may occur.

Symptoms of brain-pressure are: headache, vomiting, mental dullness, and frequently some form of paralysis with contracted pupil and finally coma.

Focal symptoms depend on the character of the lesions; if irritative, convulsive or spasmodic phenomena if located in the motor area, and, if decided pressure or destructive lesions, paralysis, such as hemiplegia and asphasia.

The symptoms of brain lesions due to the pathologic process, itself, have few if any particular symptoms other than those due to the location, except in abscess or gumma, when the constitutional symptoms of suppuration, such as chills, fever, sweats, and prostration, are added to other brain symptoms.

CONGESTION OF THE BRAIN.

Synonyms.—Cerebral hyperemia; cerebral congestion.

Definition.—An abnormal fullness of the vessels (capillaries) of the brain: *Active,* when arterial fullness; *passive,* when venous fullness; characterized by headache, vertigo, disorders of the special senses, and, if the hyperemia be decided, convulsions.

Causes.—*Active.* Increased cardiac action, the result of hypertrophy of the left ventricle; general plethora; excesses in eating and drinking; acute alcoholism; sunstroke; inhalation of amyl nitrite; prolonged mental labor; diminished amount of arterial blood in other parts; compression of the abdominal aorta or ligation of a large artery, or the suppression of an habitual bleeding hemorrhoid, are the principal causes.

Passive. Dilatation of the right heart; pressure upon the veins returning the cerebral blood; emphysema; and similar conditions interfering with the venous circulation.

Pathologic Anatomy.—The post-mortem appearances are: Overloading of the venous sinuses and of the meningeal vessels, including the finer branches; the pia mater appears vascular and opaque; the gray matter of the convolutions unduly red; the convolutions may be compressed and the ventricles contracted, with the displacement of a corresponding amount of cerebrospinal fluid. Long-continued or repeated congestions lead to enlargement and tortuosity of all the vessels, a moist and slimy condition (edema) of the cerebral substance, and an increase in the sub-arachnoid fluid.

Symptoms.—"Rush of blood to the head" may be gradual or sudden in its onset, the symptoms aggravated by the recumbent position. Headache, with paroxysmal neuralgic darts, disorders of vision and hearing, buzzing in the ears and sparks before the eyes, contracted pupils, vertigo, blunted intellect, inability to concentrate the mind, irritable temper, and curious hallucinations are present. The face is red, the eyes congested, and the carotids pulsating. The sleep is disturbed by dreams and jerkings of the limbs. If the attack be sudden (apoplectiform), unconsciousness with muscular relaxation will occur.

Cerebral hyperemia in children often presents alarming symptoms, such as great restlessness, insomnia, night-terrors, gnashing of the teeth during sleep, vomiting, contraction of pupils followed by general convulsion, etc. Any or all of these symptoms may continue more or less marked from an hour or two to a day, the child enjoying its usual health, after a sound sleep, save a feeling of fatigue.

Prognosis.—Mild cases terminate favorably in a few hours to a day or two, but show a strong tendency to recur. Severe cases (apoplectiform) may terminate in health, but usually foretell cerebral hemorrhage. The passive form is controlled by the lesions giving rise to it.

Treatment.—*Active form:* The cause should be removed if possible. Elevate the head and apply cold, either cold cloths or the ice-cap; at the same time warmth to the feet. Leeches to the mastoid, or cups to the neck, or in the apoplectiform variety venesection should be employed, to diminish the intracranial blood-pressure; compression of the carotids, or ligatures about the thighs, have been recommended.

Active purgation is indicated, either by croton oil, or magnesium sulphate, by the mouth. The following enema is often valuable:

℞. Magnesii sulphatis	℥ ij	60. Gm.
Glycerini	f℥ j	30. Cc.
Aquæ bul.	f℥ iv	120. Cc.

M. S.—Administer slowly per rectum, with little force.

In mild cases the application of an ice-cap to the head, a sinapism to the nucha, and potassium bromid, gr. xxx-xl (2-2.6 Gm.), repeated, and the enema mentioned above, control the symptoms. Fluid extract of ergot is strongly recommended, but its value seems to be overestimated.

In severe cases with forcible, overacting heart, tincture of aconite or veratrum viride may be used in addition to the measures already mentioned.

Passive form: The treatment should be directed entirely toward the condition producing the venous stasis.

CEREBRAL ANEMIA.

Definition.—An abnormal decrease in the quantity of blood in the cerebral vessels; *general*, when the diminished supply includes all the vessels; *partial*, when the diminished supply is limited in area; characterized by pallor, headache, vertigo, some loss of power, and, rarely, convulsions.

Causes.—*Partial* cerebral anemia results from obstruction of a vessel, from embolism or thrombosis. *General* cerebral anemia results from hemorrhages, wasting diseases, during convalescence from severe attacks of fevers, sudden shock, feeble cardiac action, valvular heart disease, and general anemia.

Pathologic Anatomy.—The functional activity of the brain depends upon the quantity and quality of the blood circulating in the cerebral capillaries. Any decrease in the normal quantity or impairment in the quality produces the symptoms of cerebral anemia. The brain is pale and milky in color, and on transverse section there are no bloody points; the ventricles and perivascular lymph-spaces are well filled with fluid.

In *partial* anemia the deficiency in the blood-supply is local corresponding to the area supplied by the obstructed vessel.

Symptoms.—In general cerebral anemia, there are present pallor, fainting attacks, vertigo worse on exertion, yawning tendency, headache relieved by the recumbent posture, and sometimes convulsions. In partial anemia, there is sudden loss of power of a limited muscular area which gradually returns to normal. Cerebral anemia may be acute or chronic according as to whether the causes are sudden or gradual in character.

Prognosis.—The outlook is favorable in those cases in which the cause may be removed. In cases resulting from severe and repeated hemorrhages, the prognosis is unfavorable.

Treatment.—In anemia of the brain due to general anemia, regulated diet and the administration of iron, arsenic, quinin, strychnin, etc., should be prescribed. A certain number of hours daily, in the recumbent posture is of advantage. When there is a decided tendency to attacks of swooning, quickly acting diffusible stimulants such as aromatic spirits of ammonia, Hoffman's anodyne (compound spirit of ether), nitroglycerin, etc., should be given. Amyl nitrite, cautiously administered, may be used at times. The following prescriptions will be found of value in improving the quantity and quality of the blood in these cases:

℞.	Tinct. ferri chlor. ....♏ xv	1. Cc.
	Acid. phosph. dil. ......♏ v	.3 Cc.
	Liq. arsenici chloridi ....♏ iij	.2 Cc.
	Syr. limonis ....fʒ ss	2. Cc.
	Syr. zingiberis ..q. s. ad fʒ ij	ad 8. Cc.

M. S.—Every six hours, well-diluted.

Or—

℞.	Strychninæ sulph. ......gr. j	.065 Gm.
	Quininæ sulph. ....gr. xlviij	3.1 Gm.
	Acid. hydrochlorici dil. .fʒ ij	8. Cc.
	Tinct. gentian. comp. ...f℥ iij	90. Cc.
	Tinct. card. comp. q. s ad f℥ vj	ad 180. Cc.

M. S.—Teaspoonful in water, after meals.

In those cases due to heart-disease, hemorrhages, etc., the remedial measures advised for those conditions should be instituted in addition.

CEREBRAL HEMORRHAGE.

Synonyms.—Apoplexy; "a stroke."

Definition.—The sudden rupture of a cerebral vessel and escape of blood into the cerebral tissue, causing pressure and more or less destruction of the brain-substance, characterized by sudden unconsciousness, irregular, noisy respiration, and complete muscular relaxation.

Causes.—It is a disease of the aged, seldom being encountered in individuals under forty years of age. Apoplexy early in life is usually syphilitic. Under ordinary circumstances it seems to occur most frequently in the spring and autumn. The principal cause is disease of the vessels, manifesting itself in the development of miliary aneurysms or in a chronic endarteritis with an associated cardiac hypertrophy. As contributory causes may be mentioned heredity, Bright's disease, syphilis, chronic alcoholism, and the various other affections that induce arterial degeneration. The condition may be precipitated by emotion, over-exertion, acute indigestion, acute alcoholism, and similar disturbances.

Pathologic Anatomy.—The most common locations of cerebral hemorrhages are the regions supplied by the "central arteries," the internal capsule, corpus striatum, and thalamus opticus; less common, the cerebellum; next in frequency the pons and medulla oblongata, and rarely on the convexity of the brain, termed meningeal hemorrhage.

Intracerebral hemorrhage is more common upon the right than upon the left side, and especially affects the region of the caudate nucleus, lenticular nucleus, internal capsule, and optic thalamus; and particularly the outer border of the lenticular body, which is supplied by the striate artery,—the artery of cerebral hemorrhage. These lenticular striate arteries are branches of the Sylvian artery, and have no anastomoses. When the hemorrhage is large, the blood may break into the ventricles and pass by the *iter* from the third to the fourth ventricle. A recent clot is dark in color, and in consistency a soft, grumous mass, composed of coagulated blood and brain substance in varying proportions, at whose center is the opening into the ruptured vessel. The clot excites inflammation around it, resulting in its becoming encysted, by the development of new connective-tissue from the neuroglia, and then being gradually absorbed, leaving cicatrix; or the brain-tissue around the clot softens and degenerates—localized softening.

Symptoms.—The attack may occur suddenly as an apoplectic shock or stroke, or slowly with prodromes or "warnings."

Prodromes: Headache, vertigo, transient deafness or blindness, sensation of numbness of the extremities, with local palsies, together with the constant dread of an attack.

The *attack* may begin with vomiting, followed by either partial or complete insensibility, or, suddenly, the patient becoming at once unconscious and, if standing at the time, sinking to the ground completely relaxed or, rarely, with spasmodic or convulsive movements. Respiration is slow, irregular, and noisy; during inspiration the paralysed cheek is drawn in; and during expiration puffed out. The pulse is slow and full and there is throbbing of the carotids. The face is flushed, the eyes congested and the pupils are uninfluenced by light. The temperature falls a degree or two below normal but rises within twenty-four hours to 100°-101° F. In fatal cases the temperature may rapidly rise to 106°-108° F.

The muscular system is profoundly relaxed, and the reflex movements are abolished. Involuntary urination and defecation are frequent. The head and eyes deviate in many cases, toward the affected side in the brain or from the paralyzed side. Convulsions rarely occur.

Ingravescent apoplexy begins as a mild stroke with a rapid return to consciousness and power, except, perhaps, of speech. Headache is present with some one or more local symptoms, and in a few hours to a few days consciousness gradually becomes impaired, the loss of power again occurs, and the coma deepens, the patient dying comatose.

If the unconsciousness continues longer than twenty-four hours, death is the usual termination, preceded by pale face, irregular and rapid pulse and respiration, and rise of temperature.

Reaction takes place in many cases in from one-half to three hours, consciousness gradually returning and reflex excitability slowly reviving. It is associated with headache, confusion of mind, and more or less paralysis of motion and sensation on one side of the body (*hemiplegia*). The electro-excitability of the paralyzed parts is preserved. Irritation of the motor fibers shortly induces contraction of the affected muscles (primary rigidity). Contractions later in the course of the affection are of unfavorable significance as they indicate degeneration in the motor tracts (secondary rigidity). Recovery from the attack may be delayed by inflammatory symptoms, the temperature rising to 101°-104° F., and by severe neuralgic pains and the muscle-contractions.

Localization of the lesion of a cerebral hemorrhage is of great practical importance.

Capsular hemorrhage, or hemorrhage into the internal capsule at the anterior portion around the genu (knee), where the motor fibers pass and converge, coming from the hemispheres, is frequent, causing loss of consciousness of sudden or rapid onset, hemiplegia, involving face, arm, and leg, with motor aphasia if the hemiplegia be on the right side. There is also a unilateral loss of reflex action, conjugate deviation of the eyes from the paralyzed side, and unilateral defective movement with flaccidity of the limbs.

Cortical hemorrhage gives rise to localized unilateral paralysis of the face, the arm, or the leg, with local convulsions or convulsions that have a local beginning, or profound unconsciousness.

Centrum ovale hemorrhages resemble the cortical as regards the local convulsions.

Crus-cerebri hemorrhage produces loss of consciousness with hemiplegia involving the lower half of the face and the limbs, with paralysis of the third nerve on the opposite side, or the side of the lesion. The unilateral third nerve symptoms are ptosis, external strabismus, dilatation of the pupil, and loss of accommodation for near objects. The paralysis is termed "crossed" or "alternate" hemiplegia.

Pons hemorrhage causes either general convulsions or irregular convulsions in the legs, bilateral motor paralysis, bilateral anesthesia, either contracted or dilated pupils, embarrassed respiration, repeated vomiting without nausea, and high temperature. If the hemorrhage is large, death is sudden or within a few hours, and even if small, the prognosis is unfavorable.

Ventricular hemorrhages are generally of the ingravescent variety, and are characterized by a second apoplectic seizure soon after the first, with extension of the hemiplegic symptoms, or a relaxation of the muscles, from one side to both sides of the body.

Cerebellar hemorrhages vary so greatly in the symptoms that a positive diagnosis can seldom be made.

Meningeal or *dural hemorrhage* is usually due to a trauma. Two varieties: I. *Infantile meningeal hemorrhage,* occurring during labor. II. *Extra-dural hemorrhage,* the result of direct injury to the head.

The *infantile variety* presents symptoms of irritation and compression of the cortex, such as convulsions, general or unilateral; rigidity, opisthotonos, and either hemiplegia or diplegia.

The *extra-dural variety* is almost always the result of fracture or trauma of the skull, resulting in an extravasation of blood between the dura and the skull, from the middle meningeal artery;

the hemorrhage may be on one or both sides. The symptoms may develop at once or after some days, and are those of pressure; hemiplegia, partial or complete; convulsions, impaired or absent reflexes, dilatation with loss of reaction of pupil of opposite side; and stupor, gradually deepening into coma and death.

Sequelæ.—Paralysis of the muscles of the face, tongue, body, and extremities of one side, opposite to the location of the hemorrhage, termed *unilateral paralysis,* or *right* or *left hemiplegia.*

Paralysis of both sides of the body, due to simultaneous hemorrhage on both sides, termed *bilateral hemiplegia,* or *diplegia.*

Paralysis of one side of the face and of the extremities of the opposite side, due to hemorrhage into the pons Varolii, termed *alternating* or *crossed paralysis.*

Occasionally *tonic contractions* occur in muscles long paralyzed, termed late rigidity, and constitute evidence of a secondary degeneration of the nerve fibers.

Choreic movements in paralyzed muscles are termed *post-hemiplegic chorea,* due, according to Charcot, to changes in the motor centers.

The mental powers are always more or less permanently impaired, the patient irritable and emotional, with loss of memory in varying degrees.

Diagnosis.—The diagnosis of the apoplectic seizure is often one of the most difficult questions in medicine, and yet of the greatest importance, as the treatment depends upon its accuracy. The diagnosis of the sequelæ is comparatively easy.

Alcoholic Insensibility differs from apoplexy in the following points: insensibility is not so complete, no drawing in and puffing out of one cheek with respiration, the pulse frequent instead of slow, the pupils influenced by light; upon raising both legs, no difference is apparent in allowing them to drop; the eyes and head are not turned to one side, and, lastly, the condition is ameliorated on the inhalation of ammonia. Von Wedekind's test for temulence is generally satisfactory: "By simply pressing on the supraorbital notches with a steadily increasing force one may, with certainty of success, bring an unconscious alcoholic to his senses, and thus differentiate between alcoholic and other comas."

Opium poisoning differs from apoplexy by the gradual approach of the coma, the contracted pupil, slow pulse, and quiet, slow respiration; the patient can be momentarily aroused, and the heavy stertor of apoplexy is absent.

Uremia causes a coma that closely resembles apoplexy. A history of Bright's disease and the presence of albuminuria at once clear up the case; again, uremic coma is generally preceded by convulsions; a rapid rise of temperature is present as shown by the thermometer, often 104° F. to 106° F., while to the hand the surface appears but little, if at all, above the normal; the pulse is usually weak with irregular force, the respirations averaging twenty-five to thirty per minute, and the face having a glossy appearance.

Cerebral embolism cannot always be differentiated from apoplexy. We may suspect cerebral plugging if the patient be young; if he be laboring under acute or chronic cardiac valvular trouble; if, within brief periods, several incomplete attacks have occurred before a complete comatose conditions obtains; or, if hemiplegia results with passing or slight unconsciousness; or, if the phenomena are sooner or later followed by cerebral softening, since embolism and thrombosis are the most common causes of softening.

Syncope, or fainting is of sudden onset, but, being due to a failure of the circulation, the pulse is feeble, the face pale, the respirations quiet, and the duration of unconsciousness short, all the very opposite of an apoplectic attack.

Hysteria may resemble apoplexy at times but the history, sex, and other characteristics of hysteria will serve to make a distinction.

Prognosis.—If the patient survive the immediate effects of a cerebral hemorrhage, he is always in danger of another attack, since the causes of the original attack still remain. Another attack or two is the usual course, a fatal termination ultimately occurring. If the attack be due to or associated with Bright's disease, recovery is rare. The hemiplegia is uncertain; a partial recovery may occur within a few months or it may continue for years. The symptoms to be looked upon with alarm include long-continued loss of consciousness, abolition of reflexes, respiratory disturbances, disorders of the cardiac function, etc.

Treatment.—If there are prodromal indications, the most prompt means of reducing the intracranial blood-pressure is by venesection, followed by a brisk purgative, which may be aided by an immediate enema:

℞. Magnesii sulph. ....℥ ij 60. Gm.
Glycerini ....f℥ j 30. Cc.
Aquæ bull. ....f℥ iij 90. Cc.
M. S.—Administer by bowel slowly without force.

If the patient is weak, however, leeches should be applied to the mastoid instead, and potassium bromid, gr. xl-lx (2.6-4 Gm.), or the fluid extract of ergot, fʒss-i (2-4 Cc.), should be administered.

During the attack, the clothing should be loosened and all constrictions removed. The patient should be placed in a perfectly quiet, cool room, allowing him to lie sufficiently on his side, with the face somewhat downward, in order that the tongue, palate, and secretions may fall forward instead of backward into the pharynx. The head and shoulders should be elevated slightly and an ice-bag applied to the head. A hot mustard foot-bath should be employed. Venesection should be performed at once as it aids in lessening the cerebral congestion. Prompt catharsis by means of croton oil, ♏i-iii (0.06-0.2 Cc.), with glycerin, ♏xv (1 Cc.), placed on the back of the tongue, is also advisable. If the pulse is full and strong after consciousness is regained, either tincture of veratrum viride or tincture of aconite is indicated. If, during the attack, the face becomes pallid, and the pulse irregular, and the patient is prostrated with shock, diffusible stimulants such as ammonia and ether, cautiously employed, will be of great value.

For the secondary fever, tincture of aconite or tincture of veratrum viride may be used, and for the headache and delirium camphor and bromids may be employed. Absorption of the clot may be hastened to some extent by keeping the secretions active and by the administration of potassium iodid or bichlorid of mercury alternating with:—

℞. Liq. potassii arsenit. ....♏ v .3 Cc.
Syr. calcii lacto-phosph..fʒ ij 8. Cc.
M. S.—Three times a day.

Subsequent to the attack the patient should be placed on a liquid or semisolid diet. Absolute cleanliness in the care of the patient is highly essential in order to prevent the formation of bed-sores. The bowels should be moved daily and the quantity of urinary secretion should be carefully watched. Bathing with alcohol serves to render the patient very comfortable. Frequently the speech is lost, either temporarily or permanently, and in these cases the attendant should inquire after the needs of the patient, as otherwise he may be seriously neglected. After two or three months a weak galvanic current applied directly to the head by placing an electrode on each mastoid process, promotes absorption. For the paralyzed muscles, the faradic current, applied by placing one electrode over or near the nerve innervating the muscle and the other over its belly, acts as a tonic, preventing wasting; it is assisted by hypodermic injections into the paralyzed muscles of strychnin sulphate, gr. 1-64 (0.001 Gm.), four times a week. Massage and warm salt baths are also of value.

CEREBRAL THROMBOSIS AND EMBOLISM.

Synonyms.—Partial cerebral anemia; occlusion of cerebral vessels; cerebral apoplexy (?).

Definition.—The occlusion of a cerebral vessel, from the formation of a thrombus or the presence of an embolus, thus causing anemia of some portion of the brain; characterized by gradual—when the result of thrombosis, and sudden, when due to embolism—development of headache, vertigo, disorders of intelligence, with more or less complete insensibility and paralysis.

Causes.—*Thrombosis*, or the formation of a clot in the vessel,—an ante-mortem coagulation,—is almost always the result of chronic endarteritis, as seen in the aged, together with a slowing and weakening of the blood current. Chronic alcoholism and syphilis are the usual causes when occurring in young adults.

Emboli, in the great majority of instances, result from an endocarditis—cardiac emboli; small particles of the exudation being carried into the circulation and deposited in the brain. Emboli may also be derived from an aortic aneurysm or syphiloma of the great vessels.

Pathologic Anatomy.—The cerebral arteries may be obstructed by emboli or thrombi: the cerebral veins and sinuses by thrombi only. The changes in the cerebral tissue are those of anemia of the part or parts supplied by the occluded vessels. The subsequent changes depend upon the anatomy of the vessels. If the obstructed artery has anastomoses, the collateral circulation is soon established and the brain-tissue assumes its normal condition. If, on the other hand, the occluded vessel be one of "Cohnheim's terminal arteries,"—arteries without anastomoses, such as the lenticular-optic and the lenticular-striate set of arteries, branches of the Sylvian artery,—the blood in the whole extent of the occluded vessels coagulates.

As a result of the anatomic arrangement, collateral circulation is never established and the anemic structure supplied by the affected vessel dies or undergoes necrobiosis followed by yellowish-white softening. If the vessel beyond the seat of the occlusion remains pervious, blood flows back through the capillaries from the nearest artery or vein, the parts that a short time before were bloodless, now become deeply engorged, the succeeding changes in the vessels permitting diapedesis of the red blood globules. The tissues which are undergoing disintegration are colored by the red globules, causing the appearance known as "red softening," which after some weeks becomes "yellow softening," finally changing to "white softening," when there is a milky, or rather creamy fluid mixed with masses or particles of broken-down nerve elements. Infective emboli may produce abscesses in the brain.

The vessel most commonly occluded is the *left middle cerebral artery*, which sends branches to the second and third frontal convolutions, the anterior and superior portions of the three temporal convolutions, the island of Reil, the parietal convolutions, part of the external and all of the internal capsule, the lenticular nucleus, and most of the corpus striatum,—the *motor centers*.

Symptoms.—*Thrombosis* is characterized by a gradual onset. It is most common in the aged and is manifested by persistent headache and vertigo of varying intensity; alterations in the character, the patient becoming irritable, morose, and despondent with periods of absent-mindedness; disorders of vision, impairment of memory; hesitating and mumbling speech; impaired locomotion with muscular weakness and trembling; and finally paralysis. Hemiplegia is common and may appear gradually or be preceded by sudden insensibility; the condition progresses and ends in dementia and finally death from exhaustion. Rarely, a collateral circulation is established and partial or complete recovery occurs.

In *cerebral embolism,* the symptoms occur suddenly and may be mild or grave in character.

In the mild variety, there are sudden and severe vertigo, confusion of mind, muscular twitchings, usually one-sided, and vomiting, followed by hemiplegia, most frequently of the right side, the intellect remaining clear but hesitating. After some weeks or months the paralysis usually disappears and recovery is complete.

The *grave or apoplectic variety* is manifested by sudden headache, vertigo, flushing or pallor of the face, sudden unconsciousness, often preceded by a sharp cry, and complete muscular relaxation followed by death, or a gradual return to consciousness with hemiplegia, usually right-sided, and aphasia. The loss of speech may last several weeks or months or may be persistent. The mind may remain normal or may be greatly enfeebled, the reason ànd judgment be clouded, and after a varying period dementia develops, being followed by exhaustion and death.

The following localizing signs will serve to determine the situation of the obstruction:

Vertebral artery, the left most frequently, when obstructed results in acute bulbar paralysis from involvement of the nuclei in the medulla, with or without hemiplegia.

Basilar artery obstruction causes diplegia with bulbar symptoms. There is rapid rise of temperature. Death follows within a day or two, or suddenly, if the respiratory centers are involved.

Middle cerebral artery or one of its branches is the most frequent seat of embolic or thrombotic occlusions. The symptoms depend upon the exact branch involved; if plugged before the central arteries are given off, the internal capsule is deprived of its blood-supply and permanent hemiplegia may follow; if the blocking is in the central branches, the hemiplegia involves the arm and face, and if the left side, aphasia occurs. The individual branches passing to the third frontal (aphasia), the ascending parietal (hemiplegia, particularly hand), supra-marginal and angular gyri (word blindness), and the temporal gyri (word deafness), may be plugged.

Duration.—*Thrombosis* is essentially an affection of the elderly, and has a chronic course. Months and years may be occupied with the various symptoms until the phenomena of secondary dementia develop.

Embolism is of sudden onset, and may be followed by a rapid recovery.

Diagnosis.—*Thrombosis* is associated with changes in the vessels, the arcus senilis, and other evidences of senile degeneration.

Embolism may be mistaken for cerebral apoplexy, and, while a positive differentiation cannot always be made, an important point to be considered is the presence of cardiac murmurs.

Prognosis.—*Thrombosis* is a permanent and progressive condition in the majority of instances. Recovery is a rare termination.

Embolism may be followed by a perfect recovery. Usually, however, some evidences of the plugging remain permanently. Death may be the result within a day or two, from the plugging of a large vessel, the patient never emerging from the coma. In other cases the patient arouses from the coma, the hemiplegia with aphasia persisting, and the case pursues the usual course of localized cerebral softening.

Treatment.—The indication in the early stage of embolism and thrombosis is to reëstablish the circulation within the area deprived of its blood-supply, in order to prevent the changes incident to defective nutrition; this is accomplished by measures to strengthen the heart's action, tonics, perfect rest for some time after the attack, a plain but nutritious diet, and attention to the various secretions. Bartholow advises the administration of ammonium carbonate, gr. x (0.6 Gm.), and ammonium iodid, gr. v (0.3 Gm.), three times daily over a long period, the objects being to increase the action of the heart and arteries, and to effect a solution of the thrombus by maintaining the alkalinity of the blood. Rest in bed with the head elevated, should never be neglected in these cases. In cases in which syphilis is a factor, potassium or sodium iodid and mercury should be given. Stimulants in moderate doses are of value.

In the aged, presenting indications of degeneration, much benefit results from the use of—

℞. Liquor. potassii arsenitis . ♏ iij	.2 Cc.
Syr. calcii lacto-phosphat. f ʒ ij	8. Cc.

M. S.—After meals, well-diluted.

It may be combined with cod-liver oil with decided advantage.

For embolism, the immediate and persistent use of the following may dissolve the plug:

℞.	Ammonii carbonat. .....gr. v	.3 Gm.
	Liquor. ammonii acetatis f℥ j	4. Cc.

M. S.—Three or four times daily, well-diluted.

In a month or two a very light galvanic current (from two cups) may be passed through the brain in both directions (Bartholow).

CEREBRAL ABSCESS.

Synonyms.—Acute encephalitis; suppurative encephalitis.

Definition.—An acute suppurative inflammation of the brain structure, either localized or diffused, primary or secondary; characterized by impairment of intellect, sensation, and motion.

Causes.—*Primary cerebral* abscess is exceedingly rare, and is due to pyemia, glanders, and embolus from ulcerative endocarditis.

Secondary cerebral abscesses result from injuries to the cerebral tissues, following apoplexy, embolism, thrombosis, and injuries to the cranial bones, chronic suppurative otitis, and chronic suppuration in some other portion of the body.

Pathologic Anatomy.—Abscesses of the brain may be single or multiple, varying in size from an almond to an egg.

They occupy a limited and well-defined region of the cerebral tissue, such as either the corpora striata, optic thalami, gray matter of the cortex, the cerebellum, or the white matter of the hemispheres. Cerebral abscesses are usually due to microörganisms and are more frequent in the right hemisphere than the left. When the result of pyemia or infection from distant organs, such as the lungs, they are generally multiple. When secondary to disease of the ear, frontal sinuses, naso-pharynx, or trauma, they are usually single.

An abscess having developed, steadily increases in size, encroaching upon the surrounding brain, and usually the brain-tissue forms a defensive wall about the abscess—a capsule or pyogenic membrane. The encapsulated abscess continues to develop, and finally bursting, infiltrates the surrounding tissue with consequent pressure, or discharges into the meshes of the pia-arachnoid, on the cortex, or into the lateral ventricles. Rarely, an encapsulated abscess may become permanently encysted. The pus of cerebral abscess is greenish or greenish-yellow in color and fetid (Dercum).

Symptoms.—A concise description of the symptoms of abscess of the brain is very difficult, on account of the wide variations dependent on its location, and also the difficulty of isolating it from the affections to which it is secondary.

The onset varies according to the cause, although all cases are associated with headache, irritative fever, vomiting, persistent and spreading paralysis, convulsions, optic neuritis, mental apathy, delirium, and coma.

If consecutive to apoplexy, thrombosis, or emboli, there occur fever and delirium, the paralysis remaining and spreading with spasmodic contractions of the affected muscles.

If secondary to a chronic ear-disease, there is sudden cessation of the ear-discharge; severe pain in ear and side of head, accompanied with chill, fever, vomiting, followed in a few days by the disappearance of febrile symptoms and the development of a condition of stupor, with cerebral symptoms, depending upon the location of the abscess.

Occasionally, cases run a chronic course with rather insidious onset, dull, persistent headache; changed disposition, peevish, irritable, unreliable, with decline of moral sensibility; easily fatigued by mental work; inability to stand exertion; impaired memory; vertigo; and dyspepsia; soon followed by slight palsies, which progressively increase, becoming general, with involuntary discharges, death following from exhaustion.

Of the focal symptoms, hemiplegia, of incomplete character, occurs in about one-half of all cases of abscess of the brain. A very constant symptom of diagnostic value, when hemiplegia is very marked, is exaggerated knee-jerk with pronounced ankle clonus.

Diagnosis.—A positive diagnosis is only possible by a close study of the causes and the clinical history, as the symptoms at times indicate meningitis and again cerebral tumor.

Purulent meningitis may follow trauma to the brain or chronic ear disease, making the diagnosis impossible. The chief points of distinction are: The subacute or chronic course of abscess, slight involvement of cranial nerves, hemiplegia, and the presence of an active, persistent, unilateral ankle clonus and exaggerated knee-jerk on paralyzed side.

Prognosis.—The usual termination is in death. The course depends upon the character and extent of the injury, varying from a few days to several months.

Treatment.—Surgical treatment has been attended with marked success in some cases of abscess of the brain, the withdrawal of the pus being followed by recovery. For traumatic abscess the operation of trephining is indicated. When operation is for any reason impracticable, the treatment is purely symptomatic.

INTRACRANIAL TUMOR.

Synonym.—Cerebral tumors.

Definition.—Tumor of the brain is either a growth in the cerebral tissue, on the meninges, or in the vessels, and is characterized by symptoms of pressure upon the brain-structure.

Causes.—The most important etiologic factors are male sex, middle age, heredity, injuries to the head, vascular changes, syphilis, tuberculosis, and cancer.

Pathology.—Tumors of the brain are of various kinds—to wit: *vascular tumors*—aneurysms; *parasitic tumors*—cysticercus; *diathetic tumors*—tubercle or syphilis; *accidental tumors*—glioma. The most common cerebral growths are tubercle, syphiloma, glioma, sarcoma, carcinoma, and cysts. The size of the tumor varies and they may attain the size of an orange before they induce symptoms. All growths of the brain produce irritation of the surrounding parts at first, and later by pressure give rise to interference with the blood-supply and destruction of the tissues.

Symptoms.—Those common to all cerebral tumors are: Headache, persistent and increasing in intensity; defects of vision, even blindness, due to an optic neuritis, a very constant symptom; defects of hearing, taste, and of speech, the result of paresis of the vocal cords; vertigo, associated with nausea and vomiting and convulsions, epileptiform in character. These convulsions are usually limited to one side of the body and occur at regular intervals. They may be localized (Jacksonian epilepsy) to one or more groups of muscles and are never attended with loss of consciousness. Nystagmus is sometimes present. Palsies such as strabismus, ptosis, dilatation of the pupil, facial palsy, paraplegia, and hemiplegia are not uncommon symptoms. Defects of sensibility such as sensations of numbness and coldness in the limbs and body may also occur. Occasionally, there are disturbances of equilibrium, manifested by a tendency, when standing, to go backward or turn to the right or left. The intellectual faculties may be well-preserved until late in the affection when irritability of temper, depression of spirits, impairment of memory, emotional disturbances, and a gradually advancing dementia may develop. Slowing of the pulse, insomnia, and increased secretion of urine sometimes accompany cerebral tumors.

Diagnosis.—A positive diagnosis can rarely be made. The following points will aid: Long-continued persistent headache, without appreciable cause; unilateral epileptiform convulsions, without loss of consciousness; difficulty of vision, hearing, and speech, associated with nausea and vomiting, and local and general palsies.

The location of the tumor may be determined by the more or less pronounced character of certain symptoms.

The diagnosis of the character of the growth can only be determined by a close study of the history.

According to Herter, the indications that suggest that the tumor is a *syphilitic growth* are as follows: Syphilitic history; symptoms of irritative disease of cortex rather than destructive evidences of rapid growth at the onset, followed by a period of slow progress or stationary symptoms; gradual improvement under antisyphilitic treatment; development between twenty and forty-five years of age.

Indications suggesting *tubercular growth* are: Family history, or tuberculosis in some other organ of the patient; rapid development of symptoms; indications of the growth in the cerebellum or in the pons; early appearance of the symptoms, especially before the tenth year, and history of injury to head.

Indications suggesting *sarcoma* or *cancer* are: The presence of a sarcoma elsewhere and rapidly failing health, with cerebral-tumor symptoms in patient over fifty years.

Indications suggesting *glioma:* Sudden loss of consciousness with exacerbation of all symptoms in the clinical history of cerebral tumor; cortex irritative symptoms as in syphiloma, developing under fifty years of age, and the absence of all evidences of tubercle, syphilis, sarcoma, and cancer.

The *focal* symptoms of intracranial tumors are so important in diagnosis that the following summary of symptoms caused by brain tumors is given:

Prefrontal region. Mental impairment; pressure in central region, causing aphasia, Jacksonian epilepsy, and disturbances of smell.

Central region. Motor aphasia, monoplegia, partial anesthesia, Jacksonian epilepsy.

Posterior parietal region. Word-blindness, homonymous hemianopsia, disturbed muscular-sense.

Corpus callosum. Progressive hemiplegia.

Crus cerebri. Crossed paralyses of oculo-motor nerve and limbs.

Corpora quadrigemina. Oculo-motor paralyses, reeling gait, possibly blindness and deafness.

Pons and medulla. Crossed paralysis of face and limbs, or tongue and limbs. Other lesions in cranial nerves.

Cerebellum. Marked cerebellar ataxia, vomiting, convulsions, coma.

Base, anterior fossa. Mental enfeeblement, disturbances of smell and vision, and exophthalmos.

Base, middle fossa. Impairment of vision; hemiplegia; oculomotor disturbances.

Base, posterior fossa. Trigeminal neuralgia; neuro-paralytic ophthalmia; paralysis of the face and tongue; impaired hearing; crossed paralyses.

Diagnosis between cerebral tumor and abscess. Both may have any or all of the following symptoms: Headache, vomiting, double optic neuritis, and mental failure. Tumor has, in addition, marked *focal* symptoms, monoplegia, hemiplegia, paralysis of cranial nerves, and marked optic neuritis; the absence of these favors abscess. If the hemiplegia is due to abscess, the ankle clonus and knee-jerk are exaggerated. Fever and rigors point to abscess. The causes of abscess are very clear, those of tumor often uncertain.

Prognosis.—Except in those cases of syphilitic origin the prognosis is very unfavorable and even in syphiloma the termination may be fatal if the treatment is not prompt.

Treatment.—This is unsatisfactory and consists largely in measures for the relief of the symptoms. As benefit occasionally follows the use of potassium iodid, gr. xx (1.3 Gm.), three times daily, and also fluid extract of ergot, f℥ss-i (2-4 Cc.), three times a day, increased until their full physiologic effects are produced, these remedies should be used in all cases, discontinuing them if no benefit follows a prolonged trial. When the evidences of syphilis are unmistakable, the mercurials should be given, in addition, pushed to their point of tolerance. When the tumor can be localized and is accessible, surgical intervention is indicated.

APHASIA.

Definition.—The loss, partial or complete, of the power of expression or comprehension of language. A loss of memory for words. Aphasia is a symptom and not a disease.

Amnesic aphasia, or loss of the memory of words by which ideas are expressed.

Ataxic aphasia, the inability to combine the different parts of the vocal apparatus for vocal expression, although the memory of words still remains, so that the afflicted person can write his ideas intelligently.

Agraphia, the inability to recognize and make signs by which ideas are communicated in written language.

Amnesic agraphia, the inability to coördinate the muscular apparatus.

Paraphasia, the mental state in which the wrong words are used to express an idea.

Paragraphia, the state in which wrong or meaningless written signs are used to express an idea.

Pathologic Anatomy.—Aphasia is not the result of any one specific lesion, but occurs during the course of several—to wit: Occlusion of certain cerebral vessels; cerebral hemorrhage; cerebral abscess or softening; meningitis; tumors; mental or moral causes; hysteria.

It is now almost definitely determined that lesions of the left middle cerebral artery, island of Reil, third frontal convolution, and parts of the corpus striatum are associated in the production of aphasia. The lesions are usually upon the left side of the brain, the aphasia being often associated with right hemiplegia.

Symptoms.—The degree to which articulate language is impaired varies from the loss of a few words to complete inability to communicate ideas. The intellect does not suffer in proportion to the loss of words; for, showing the individual an article, while he may miscall it, if it is called by name he will recognize it. This inability to convey thoughts is a source of great mental suffering, in some leading to a suicidal tendency.

A strange clinical fact is the strong tendency to profanity shown by aphasic patients.

Diagnosis.—*Aphonia,* or loss of voice, should not be confounded with aphasia, or the inability to remember words.

Paralysis of the tongue, or inability to move this organ, thereby interfering with articulate language, should not be confounded with aphasia, which, as a rule, is not associated with paralysis of the tongue.

Prognosis.—The outlook is controlled entirely by the cause. If the result of congestion of the brain or a syphilitic tumor, the prognosis is favorable. If associated with hemiplegia, the clot may undergo absorption, and recovery follow. If associated with softening of the brain, however, the disease grows progressively worse.

Treatment.—The cause must be energetically treated, as the aphasia pursues a course parallel to the associated malady. Cases not associated with cerebral softening have regained the memory of words by a course of caefully conducted speech lessons.

When the aphasia is of sudden occurrence it is strongly significant of injury to the brain by a spicule of bone or the pressure of a blood-clot particularly in those cases in which there is a history of a head-wound. In these instances, the operation of trephining may be of benefit and should be considered.

VERTIGO.

Synonym.—Dizziness.

Definition.—Vertigo, or dizziness, is a subjective state, in which the individual affected (subjective vertigo), or the objects about him (objective vertigo), seem to be in rapid motion, either of a rotary, circular, or to-and-fro character.

Causes.—The etiology of an attack of vertigo depends upon the particular variety.

Ocular vertigo results from the paresis of one or more of the ocular muscles, eye-strain, or astigmatism.

Aural or auditory vertigo, or *Ménière's* disease, results from disease of the semicircular canals and cochlea. Ménière's disease, so called, is a sudden severe vertigo, the result of either a hemorrhage or a serous or purulent exudation into the semicircular canals.

Gastric vertigo is the most common variety, and results from either stomachic or intestinal dyspepsia, disordered hepatic function, or constipation. "The mechanism of the vertigo is complex. There are two factors: one consists in the toxic effect of the imperfectly oxidized materials which accumulate in the blood; the other is reflex. An impression made on the end organs of the pneumogastric in the stomach is reflected over the sympathetic ganglia" (**Bartholow**).

Nervous vertigo is associated with migraine, sick or nervous headache, and may be caused by physical or nervous excesses, and also by the immoderate use of tea, coffee, alcohol, and tobacco. It is also a result of many of the organic diseases of the brain.

Senile vertigo is the result of the disordered cerebral circulation resulting from senile changes in the heart and vessels.

Symptoms.—In all varieties of vertigo, the symptom of a *sensation of objects moving around the patient*, or *the patient moving around objects which remain stationary*, is present in some degree. The attack of giddiness comes on suddenly, with an indistinctness of vision and slight confusion of the thoughts. The patient may fall unless he grasps something to steady himself. Nausea and vomiting and cardiac palpitation with tinnitus aurium are often associated with the vertiginous sensations. *There is no loss of consciousness.*

In the ocular vertigo, the attack is usually the result of reading, writing, sewing, or other close application of the eyes, the ordinary symptoms of vertigo being preceded by headache, nausea, specks before the eyes, and pain in the eyeballs.

In *Ménière's disease,* the vertigo is associated with serious tinnitus aurium, and the vertiginous sensations are of various forms such as a see-saw movement, a gyratory motion, right or left; a vertical whirl, or a sensation of rising and falling like unto the swell of the ocean. The symptoms are of long duration, becoming marked in paroxysms. The attack of aggravated vertigo is so sudden and overwhelming at times, that the person is suddenly thrown to the ground as if struck with a blow, and is associated with nausea and vomiting. As the condition continues, the character of the individual changes, becoming morose, irritable, and suspicious. Not all cases of Ménière's disease become permanent; it may occur in isolated attacks, the interval being free from all sensations.

Gastric vertigo is by far the most frequent variety. Persons subject to vertigo of this kind live in constant dread of cerebral disease, which fear frequently results in true melancholia.

The vertiginous sensations usually occur during the course of well-marked and long-standing stomach and intestinal disorders, such as pain or oppression after meals, nausea, pyrosis, heartburn, frequent eructations, and constipation or, rarely, diarrhea. The abdomen is often distended with flatus. Great pain in the nucha is a very frequent occurrence. The attack may be associated with either hyperemia or anemia of the brain. The symptoms are not constant, but recur at intervals, sometimes remote, at others very close to each other.

In *nervous vertigo* the vertiginous symptoms are usually associated with more or less irritability of temper, restlessness, and insomnia. The onset is sudden, after some one of the etiologic factors. In megrim there are headache, nausea, and vomiting. This form of vertigo often precedes or replaces the epileptic convulsion. And it also often precedes softening of the brain.

In *senile vertigo* the vertiginous symptoms are the result of anemia of the brain. The attacks are developed by an exertion, often by merely assuming the erect posture. There is a swimming sensation in the head, and darkness falls on the eyes, with a sensation of chilliness and prostration.

Diagnosis.—The diagnosis of the various forms of vertigo can only be determined after a close study of the history and course of the attack. The existence of organic cerebral disease must always be kept in mind in solving any case.

Prognosis.—This will be influenced by the variety of the vertigo. The prognosis is favorable in ocular and gastric vertigo. Unless the result of organic disease, the prognosis is good in nervous vertigo. In auricular vertigo the prognosis is fair, but in genuine Ménière's disease the prognosis is unfavorable, as it also is in senile vertigo.

Treatment.—In all persistent cases, the eyes should be examined under the influence of a cycloplegic and the state of refraction and muscle-balance carefully ascertained. Correcting lenses should be ordered for the most trivial ametropic condition under such circumstances, and their adjustment should receive careful attention.

When the vertigo is that of Ménière's disease, rest in the recumbent posture and the administration of quinin sulphate, gr. x-xv (0.6-1 Gm.), daily, until cinchonism is produced is advised (Charcot). In cases of syphilitic origin the iodids may be employed. Potassium bromid and the salicylates are at times of value.

In gastric vertigo, the diet should be carefully regulated. At the beginning of the treatment it is often of great advantage to place the patient on an exclusively milk diet, gradually widening the variety as improvement occurs. In these cases a course of arsenic is often serviceable. If the digestion be torpid, the tincture of nux vomica is indicated. If the bowels are constipated, benefit is obtained from fluid extract of cascara.

℞. Ext. cascaræ sagr. fld.	f℥ j	30. Cc.
Glycerini	f℥ j	30. Cc.
Tinct. card. comp.	f℥ ss	15. Cc.
Aquæ menthæ pip.	f℥ ss	15. Cc.

M. S.—One teaspoonful three times daily, well-diluted.

For nervous vertigo, the exciting cause should, if possible, be removed and such remedies as iron, quinin, and strychnin, either alone or in various combinations, should be administered. Many of these cases can be traced to the other causes of vertigo and consequently the treatment is subject to many and great variations.

For senile vertigo, a highly nutritious diet with the judicious use of whiskey is indicated. Other tonics, particularly bichlorid of mercury, arsenic, nux vomica, and nitroglycerin are of value. The tendency toward atonic dyspepsia, flatulency, and constipation in the aged should be avoided by the use of appropriate drugs and other therapeutic measures. The possibility of uncorrected presbyopia as a cause in these cases should be remembered.

In all varieties of vertigo, the patient should abstain from tea, coffee, tobacco, highly-seasoned foods, malt liquors, and alcohol, unless especially indicated.

MIGRAINE.

Synonyms.—Megrim; hemicrania; sick headache; bilious headache; blind headache.

Definition.—A unilateral paroxysmal pain in the head, accompanied by nausea, often vomiting, intolerance of light and sound and incapability of mental exertion, the brain being temporarily prostrated and disturbed.

Causes.—In the majority of patients, the nervous predisposition to migraine is inherited, but whether inherited or acquired, it commonly develops about puberty. It is more common in women than in men.

Among the many exciting causes are disturbances of digestion, irritation of the ovaries or uterus, worry, anemia, exacting mental labor, sexual excesses and insufficient sleep, and eyestrain.* The causes of many attacks, however, are wrapped in mystery, as with the best of care the attacks seem to have a periodic course.

Symptoms.—Attacks of migraine occur in irregular paroxysms, the intervals between being free from pain or nervous disturbance. For a day or two preceding the paroxysm it may be ascertained that there were a feeling of fatigue or mental depression without apparent cause, heaviness over the eyes, flatulency and indigestion.

The attack proper is ushered in by chilliness, yawning, nausea, often vomiting, and general muscular soreness, with intolerance of light, flashes before the eyes, often phantasms, noises in the ears, incapability for mental exertion, vertigo, and pain of a sharp, shooting character, of great intensity and persistency, localized most frequently in either the frontal, temporal, or occipital regions of the left side; at the same time there is tenderness over the whole side of the head. Rarely the pain is felt on the right side, and still more rarely on both sides at the same time. The nausea and other digestive symptoms may follow the onset of the pain instead of preceding it.

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*For an interesting account of the history of migraine and its relation to eyestrain the reader is referred to a paper entitled: "The History and Etiology of Migraine," by G. M. Gould, Phila., Jr. A. M. A. Jan. 16-23, 1904.

There is more or less disturbance of the circulation, temperature, and secretions of the painful parts. At times there is a marked contraction of the vessels, with the face pale, the eyes shrunken, and the pupils dilated; again, the vessels may be dilated, when the face is flushed, the conjunctivæ injected, and the pupils contracted. Motion, sound, and light aggravate the acute suffering. The urine before, during, and after a paroxysm is concentrated, and it may be that the excretion of uric acid is associated with the etiology of migraine.

The attack may continue with more or less intensity from a few hours to two or three days, the average duration being twenty-four hours.

Diagnosis.—The symptoms are so characteristic that an error seems impossible. It may, however, be confounded with anemic headache, hyperemic headache, dyspeptic or bilious headache, and neuralgic or rheumatic headache. The pains of organic brain disease must be excluded.

Prognosis.—While few cases of true migraine are permanently cured, the affection is free from danger to life. In a fair number of cases the susceptibility to attacks declines as the person advances in years, it being rarely seen after fifty years. According to Herter, cases of migraine of the ophthalmic variety appear to be not rarely followed by general paralysis of the insane. When, however, appropriate treatment is instituted in cases due to eyestrain, the response is very prompt and the affection disappears almost immediately and does not recur.

Treatment.—To abort an attack of migraine, or to dispel a paroxysm after its onset, rest in bed in a quiet darkened room, absolute diet, and the administration of morphin sulphate, gr. ¼ (0.016 Gm.), with atropin sulphate, gr. 1-120 (0.00054 Gm.), hypodermically, antipyrin, gr. xx (1.3 Gm.), or phenacetin, gr. x (0.6 Gm.), should be advised. The following combination frequently relieves the paroxysm:

℞.	Phenacetin	gr. xx	1.3 Gm.
	Caffein. citrat.	gr. v	.3 Gm.
	Camphoræ monobrom ...	gr. xx	1.3 Gm.

M. Ft. capsule No. x.

S.—One every two hours until relief.

In many attacks, fluid extract of cannibis Indica, ♏ii-iii (0.12-0.2 Cc.), every half hour or hour for a number of doses, alone or combined with fluid extract of gelsemium, in the same dose, is curative. When the attacks are associated with contraction of the vessels, the following is of value:

℞.	Potassii bromid.	gr. xxx	2. Gm.
	Morphinæ sulph.	gr. ¼	.016 Gm.
	vel		
	Codeinæ sulph.	gr. j	.065 Gm.
	vel		
	Tr. opii deodorat.	♏xxx	2. Cc.
	Aquæ menth. pip. ...	ad f℥ ss	ad 15. Cc.

M. S.—One dose. To be repeated as the occasion requires.

Locally, the application of menthol pencils to the seat of the pain is beneficial. The inhalation of spirits of camphor may at times afford relief.

During the interval, a careful investigation should be made to determine the underlying cause and its removal should then be accomplished. As most individuals possess ametropia in varying degrees, the eyestrain factor in the etiology should be immediately eliminated by proper examination of the eyes under cycloplegia and the prescribing of correcting lenses in all cases. Frequently this will be sufficient. It may be added, that not uncommonly the instillation of a cyclopegic, such as homatropin or atropin, will relieve an attack. Such drugs are contraindicated, if presbyopia is present but it should also be remembered that migraine is rather infrequent in persons past forty-five years of age.

The gastrointestinal tract is responsible for a large number of cases and quite often the individual paroxysms may be traced directly to some dietetic indiscretion. Such cases require careful regulation of the diet eliminating substances which are known to disagree with the patient. The use of tea, coffee, alcohol, and tobacco should be reduced to a minimum. Constipation should always be avoided. In all cases, extract of cannabis Indica, gr. ¼ (0.016 Gm.), three times daily for several months is of value.

When there is any apparent anemia, tonics such as iron, quinin, strychnin, and arsenic should be prescribed together with good food, fresh air, and regulated exercise.

When the disposition to the malady is inherited, Bartholow recommends the avoidance of all those conditions that tend to develop an abnormal excitability of the nervous system in addition to attention to the personal hygiene of the patient. He advises galvanization of the superior ganglia of the sympathetic; the positive pole over the ganglion and the negative on the epigastrium in the tetanic (contraction of vessels) form, and the poles reversed in the paralytic (dilatation of vessels) form.

ALCOHOLISM.

Varieties.—Acute alcoholism; chronic alcoholism.

Synonyms.—*Acute variety,* temulentia; mania-a-potu. drunk.

Chronic variety, delirium tremens; dipsomania, or oinomania.

It would hardly be correct to consider these terms interchangeable; they are rather names applied to various conditions due to acute or chronic alcoholic poisoning.

Definition.—Alcoholism is the term used to designate the physical and mental phenomena induced by the use of alcohol. Alcohol is a poison, but becomes a much more dangerous one when associated with the various toxic products which are added to flavor it.

Temulentia refers to drunkenness, or alcoholic intoxication; *mania-a-potu* is an acute mental derangement, occurring in alcoholics of strong neurotic tendencies; *delirium tremens* is an attack of delirium associated with tremors in persons with the numerous changes resulting from chronic alcoholism. Delirium tremens frequently results in alcoholics having one of the forms of nephritis, preventing the elimination of some poison developed from the ingested alcohol. *Dipsomania, or oinomania,* is an alcoholic insanity in which an individual at longer or shorter intervals has paroxysms of alcoholic desires, between which he neither wishes nor craves alcohol. {periodic sprees}

Causes.—The predisposing causes are influences arising from unfavorable moral, social, and personal conditions, and heredity.

The exciting cause is immoderate use of alcoholic beverages, of which there are three groups: 1, spirits, or distilled liquors; 2, wines, or fermented liquors, and 3, malt liquors.

Pathologic Anatomy.—*Acute Alcoholism.* The brain is the seat of an active hyperemia; the mucous membrane of the stomach and duodenum is markedly injected and covered with a ropy mucus slightly tinged with blood, and the gastric juice is altered in quality and quantity. The kidneys are also the seat of an active hyperemia.

Chronic Alcoholism. In this condition there are no organs or tissues which do not present morbid changes. The gastrointestinal mucous membrane presents the changes of chronic catarrhal inflammation; the liver, the first organ to receive the poison after the stomach, presents the changes of congestion, cirrhosis, or fatty degeneration; the kidneys show chronic congestion and often the changes incident to chronic interstitial nephritis. The post-mortem results found in twenty-five cases of delirium tremens dying in the Philadelphia Hospital showed fourteen with the changes of interstitial nephritis, eight with chronic parenchymatous nephritis, and three with fatty kidney; all showed chronic gastric catarrh and changes in the myocardium and the arteries of the heart and brain, and the aorta. The muscular structure of the heart may undergo fatty degeneration and the vessels the changes of senility. The brain-structure presents the changes of sclerosis in various stages, and there may be chronic meningitis and pachymeningitis with hematoma. The nerves are altered, atrophied, and hardened, and the neuroglia, vessels, and ganglion cells of the spinal cord show similar changes.

Symptoms.—*Acute alcoholism,* resulting from the use of a large quantity of alcoholic fluid, occurs with the symptoms varying from mild intoxication to drunkenness, passing to acute delirium and acute coma. The condition begins with a period of exhilaration, passing to semi-delirium and ending in an acute coma, when the breathing is stertorous, the face bloated and congested, the lips swollen and purplish, the pupils contracted or dilated, the pulse feeble and slow, the skin cold and clammy, the temperature depressed, and frequently control of sphincters lost. An individual so affected is said to be "*dead drunk.*"

Cases of ordinary drunkenness do not often pass beyond the stage of exhilaration, ending in a mild coma or sleep.

Mania-a-potu, or acute alcoholic delirium, is the direct result of alcoholic excess in those engaged in a sudden debauch, or who have drunk alcoholic beverages very "hard" for a comparatively short period. The individuals grow more and more excitable, lose all desire for food, are unable to sleep, become the prey of horrible hallucinations,—"the horrors,"—finally terminating in mania which resembles delirium tremens in all save the tremor, which is absent.

Delirium Tremens. In the majority of instances, delirium results in a chronic drinker, from a prolonged debauch, with abstinence from proper food. It begins by an increased tremor, insomnia, irritable, excited manner, followed by the characteristic hallucinations and illusions, during which snakes and other forms of repulsive reptiles are seen, causing the most intense horror and abject fear; it is a busy delirium, the patient being unable to remain quiet. There also occur illusions of smell and hearing. This marked excitement is followed by great depression, the skin is cold and clammy, the pulse feeble, the muscular system weak, the mind in a condition of coma-vigil, and if continuted, a febrile condition, typhoid in character, with stupor or coma, develops. Uremic symptoms frequently complicate the condition, the temperature suddenly bounding to 103° F., to 104° F., or 105° F., with albumin and casts in the scanty urine.

The ordinary duration of an attack of delirium tremens is about two weeks in those who recover, although death may occur at any time from cardiac failure, uremia, or alcoholic pneumonia. Indeed, patients sometimes die suddenly after the beginning of apparent improvement. Convalescence dates from the beginning of refreshing sleep, the patient awakening with a clear mind and desire for food. Should the delirium subside, but the patient continue to mutter and pick at the bed-clothing, the tongue becomes dry and cracked, and the regurgitation of dark brownish and bilious matter occur, the condition is critical and an early fatal termination may be expected.

Dipsomania, or oinomania, is the inherited or acquired mental condition which craves the drinking of intoxicating liquors. This is a true mental disease. It manifests itself in periodic attacks of excessive indulgence in alcoholic drinking, or this symptom of the sad disease may be replaced by other irresistible desires of an impulse kind, such as lead to the commission and repetition of various crimes, the gratification of other depraved appetites, robbery, or even homicide. Imbecility and dementia frequently result.

The paroxysms at first occur at long intervals, but gradually the intervals become shorter and shorter until the individual entirely surrenders himself to alcoholic and other excesses.

Chronic Alcoholism. The condition to which this term has been given is truly a disease. It is the result of the continued use of alcoholic beverages until one or more of the morbid organic changes have occurred. These persons are markedly dyspeptic, with coated tongue, fetid breath, and early morning vomiting, straining, or retching, attended with much distress. There is a gradually developing muscular tremor, progressing to the ataxic gait. Insomnia or restless sleep is frequent. The face may either become pallid, flabby, and bloated, with an imbecile expression, or swollen, rough, and dusky, with great bladders under the eyes, and yellow, injected conjunctivæ. There are headache, vertigo, and attacks of hallucinations; the memory grows weaker, the judgment less accurate, the moral sense blunted, and the will power weak and erratic. These and many other symptoms add to the distress of the individual, which he attempts to overcome by the use of more and more of the poison.

Diagnosis.—Profound drunkenness, or alcoholic coma, may and often is confounded with apoplectic and uremic coma. Von Wedekind suggests the following method for diagnosing drunkenness: "By simply pressing on the supraorbital notches with a steadily increasing force one may, with certainty of success, bring an unconscious alcoholic to his senses, and thus differentiate between alcoholic and other comas."

The symptoms of chronic alcoholism often bear a close resemblance to the following maladies: General paralysis, disseminated sclerosis, paralysis agitans, locomotor ataxia, cerebral and spinal softening, epilepsy, dementia chronica, and nervous dyspepsia.

In individuals whose habits are secret, the question of diagnosis is attended with considerable difficulty. Anstie lays much stress upon the importance of the following four points, diagnostic of chronic alcoholism: *insomnia, morning vomiting, muscular tremor, and causeless mental restlessness.*

Prognosis.—In acute alcoholism, the prognosis is good if the patient is manageable.

In chronic alcoholism, the organic changes, the direct result of the alcoholic habit, tend to shorten life by the production of fatty heart, Bright's disease, insanity, impotence, epilepsy, melancholia, and organic brain diseases. The danger in delirium tremens is heart-failure or deepening coma. The association of chronic nephritis with delirium tremens, perhaps its cause, must always be taken into account in determining a prognosis. Acute lobar pneumonia is a very fatal complication in all forms of alcoholism.

Treatment.—In deciding upon a plan of medication for any of the varieties of alcoholism, the condition of the kidneys, heart, and vessels must be considered. The treatment of a case of ordinary drunkenness requires little consideration, as the rapid elimination of the alcohol soon occurs if its ingestion be stopped. The contents of the stomach should be immediately removed by the stomach-tube or by the hypodermic injection of apomorphin, gr. 1-10 (0.0066 Gm.). If the attack is not sufficiently severe to warrant these procedures, fractional doses of calomel every half hour followed by a saline will be of great benefit. The solution of ammonium acetate in large, frequently repeated doses greatly assists in the elimination of the poison. When the excitement is extreme, chloral, gr. xv-xxx (1 to 2 Gm.), should be given. Morphin is of great value in many of these cases but the presence of any kidney complication is a contraindication for its use. Aromatic spirits of ammonia is also of value in this condition.

For the collapse following a lethal dose of alcohol, the stomach should be immediately emptied by emetics or the stomach-tube or pump, and the organ washed out with warm water or coffee, the patient placed in the recumbent position, and surrounded with artificial warmth, hot frictions to the lower extremities. Resort should be had to artificial respiration or the use of faradism to the thorax, inhalations of ammonia, and hypodermic injections of strychnin sulphate, nitroglycerin, digitalis, strophanthus, or atropin sulphate. Tapping of the precordial region with a hot spoon (Corrigan's hammer) may serve also to stimulate the flagging heart.

For *mania-a-potu*, the immediate and complete withholding of alcoholic beverages is essential for its successful treatment. The patient should be quieted as soon as possible. The restlessness, insomnia, delirium, and visual and auditory hallucinations are usually controlled with chloral, and, on account of the gastric torpor and catarrh, interfering with the prompt absorption of medicaments, is best given by the bowel:

℞. Chloral	gr. xx-xxx	1.3-2 Gm.
Infus. digitalis	f℥ j	30. Cc.

M. S.—Repeat in two hours, in milk.

If for any reason an enema is impracticable, chloral or trional, gr. xxx (2 Gm.), should be given by the mouth, or hypodermic injections of morphin sulphate, gr. ¼-⅓ (0.016-0.022 Gm.), combined with atropin sulphate, gr. 1-100 (0.00065 Gm.), or hyoscin hydrobromate, gr. 1-100 (0.00065 Gm.), may be employed. Chloralose, gr. v-x (0.33-0.66 Gm.), and paraldehyd, fʒss-i (2-4 Cc.), may also be used. Physical restraint may be required. An attack of acute alcoholism, or mania-a-potu, may often be aborted with trional, gr. xxx (2 Gm.), repeated in two hours, or chloralamid, gr. xxx-xl (2-2.6 Gm.), repeated. Excellent and prompt results follow the use of a hot-air bath until profuse sweating occurs.

If one or two medicinal doses of the selected sedative drug do not produce quiet and sleep, be most cautious in repeating, remembering that the patient is suffering from the depressing effects of a cardiac and nerve poison, which is best combated by eliminating action on skin, bowels, and kidneys, and the administration of food. If the attack be associated with symptoms of cardiac depression, resort to brisk friction, hot alcohol and water sponging, artificial warmth, stimulating enemata, and the hypodermic administration of strychnin sulphate, gr. 1-29 - 1-30 (0.003-0.002 Gm.), repeated, or caffein citrate, gr. iij (0.2 Gm.), or digitalis.

The general nutrition should be given attention as in most cases it will be found that the patient has had very little food for several days. If the stomach will tolerate food,—and usually it will,—milk diluted with liquor calcis or Seltzer water, or hot beef tea, strongly seasoned with capsicum, should be administered every hour or two in small amounts

The appetite is stimulated by the use of the following:

℞.	Tinct. nucis vomicæ	fʒ iv	15. Cc.
	Tinct. capsici	fʒ iv	15. Cc.
	Tinct. cinchonæ comp. ..	f℥ ij	60. Cc.

M. S.—One teaspoonful, diluted, every two or three hours.

This stomachic stimulant may be alternated with aromatic spirits of ammonia, fʒj (4 Cc.), given in hot milk, with advantage to the heart and nervous system. The bowels should be moved at once by the administration of an enema:

℞.	Magnesii sulph.	℥ ij	60. Gm.
	Glycerini	f℥ j	30. Cc.
	Aquæ bul.	f℥ iv	120. Cc.

M. S.—Use as directed.

The kidneys should be stimulated by full doses of spirits of nitrous ether if the patient is able to swallow, and if not, by the hypodermic injection of caffein citrate.

In *delirium tremens,* the patient should be isolated and placed under the care of a skillful sensible nurse. The alcohol may be entirely withdrawn or its quantity greatly reduced. Tyson advises complete withdrawal of the poison, combatting any resulting adynamia with ammonia, digitalis, and strychnin. The stomach should be washed out daily and an easily digested diet should be supplied. A free action of the skin, kidneys, and bowels should be obtained as soon as possible to effect elimination of the poisonous products retained in the system. The excitability of the nervous system should be controlled by nerve sedatives. For this purpose, hypodermic injections of morphin sulphate, gr. ¼ (0.016 Gm.), combined with atropin sulphate, gr. 1-100 (0.00065 Gm.), or hyoscin hydrobromate, gr. 1-100 (0.00065 Gm.), or chloral or trional by the mouth or rectum are especially applicable. When the stomach is not too irritable, the following will be found of value:

℞.	Chloral	ʒ iv	15. Gm.
	Tr. capsici	fʒ ij	8. Cc.
	Aquæ menth. pip. q. s. ad	f℥ vj	ad 180. Cc.

M. S.—Tablespoonful every two hours until sleep, alternated with a cup of hot beef-tea, to which has been added a bolus of capsicum, gr. xx (1.3 Gm.).

Care should be taken not to produce coma by these remedies. Not more than two doses in six hours should be allowed but instead push the administration of hot liquid diet and atropin sulphate, gr. 1-64 (0.001 Gm.), with strychnin nitrate, gr. 1-32 (0.002 Gm.), hypodermically, as experience has proven that these drugs given three times daily in reducing doses, are the physiologic antidotes to the alcoholic toxin.

When the depression and cardiac weakness are great, strychnin sulphate, caffein citrate, spirits of chloroform, ammonium carbonate, strophanthus, and digitalis are of value. Atony of the stomach requires lavage and the administration of the previously mentioned capsicum mixture. When for any reason the nerve-sedatives already advised are contraindicated, paraldehyd, chloralamid, or the bromids may be employed. Strict attention must be given, at all times, to the condition of the skin, bowels, and kidneys. If the heart is not much depressed, the cautious use of the hot-air bath or the hypodermic injection of pilocarpin hydrochlorate, gr. ⅓ (0.02 Gm.), repeated at the onset of the mania will be found of great value.

Chronic Alcoholism.—The combination of symptoms termed chronic alcoholism, are the direct result of the continuous action of a toxic principle, and no success of even a temporary kind, can be expected unless the poison be withdrawn. The rapidity with which this can be accomplished is a question for the skill, judgment, and experience of the physician to determine; the chief obstacle to its success will be found to be moral rather than physical. Next to the disuse of alcohol is the question of diet. Progress will be made as the appetite and digestion improve, and great attention should be given to these. The general health will also be benefited by fresh air, exercise, mental occupation, and cold or tepid sponging and an occasional hot bath at bedtime. For the combination of symptoms of spirit-craving, morning vomiting, muscular tremor, mental restlessness, and insomnia, no drug is comparable with strychnin nitrate, either hypodermically twice daily, or, what is preferable, by the stomach to secure its local action on the mucous membrane. If the insomnia be persistent in spite of the foregoing treatment, the temporary use may be made of such remedies as chloral, morphin sulphate, paraldehyde, or trional. In many cases it is desirable, for its mental effect, if no other, to administer what the patient terms a substitute for his alcoholic beverages. The following is a good combination for that purpose:

℞.	Tincturæ nucis vomicæ. .f℥ ss	15. Cc.
	Tincturæ capsici ....f℥ ss	15. Cc.
	Ext. lupulini fld. ....f℥ iij	90. Cc.
	Inf. gent. comp. ....f℥ ij	60. Cc.

M. S.—Dessertspoonful three or four times daily, well-diluted.

For the anemia, loss of strength, and mental debility, benefit may follow the use of syrup of hypophosphites with strychnin.

Dipsomania.—The management of these cases is much the same as has already been mentioned for chronic alcoholism, although the strychnin sulphate treatment should be given the preference.

HEAT STROKE.

Synonyms.—Insolation; sunstroke; thermic fever; coup-de-soleil; heat exhaustion.

Definition.—A depression of the vital powers, the result of exposure to excessive heat. The condition manifests itself as acute meningitis (rare), heat exhaustion (common), and as true sunstroke.

Causes.—Exposure to the influence of excessive heat, either to the direct rays of the sun or artificial heat in confined quarters, or diffused atmospheric heat without proper ventilation.

Among the predisposing causes which act by lessening the power of the system to resist the heat, are great bodily fatigue, overcrowding, and intemperance.

Pathologic Anatomy.—The action of the heat upon the system is so sudden, and the malady so rapid in its course, that structural changes seldom develop. The left ventricle is firmly contracted (Wood). The right heart and vessels are engorged with dark fluid blood. All the tissues and organs of the body are in a state of great venous congestion. The blood is dark, thin, and either feebly alkaline or decidedly acid, and its power of coagulability is destroyed. The post-mortem rigidity is early and marked.

Symptoms.—These depend upon the variety of the affection.

Acute meningitis, the result of exposure to heat, has symptoms similar to those of cases due to other causes.

Heat exhaustion develops with a rapid feeling of weakness and prostration; the surface is cool, the face pale, the voice weak, the pulse rapid and feeble, the respiration increased, the vision grows dim and indistinct, noises develop in the ears, the individual, overcome, becomes partially or completely unconscious. In some cases the attack of prostration is sudden, the person falling unconscious, with perhaps convulsions or tremors, and shrunken features.

Sunstroke develops suddenly, with or without prodromes, and is manifested by insensibility with or without delirium, convulsions, or paralysis, flushed and hot body-surface, injected conjunctivæ, rapid and shallow or labored and stertorous breathing, quick pulse either bounding or weak, and an axillary temperature ranging from 105° to 108° to 110° F., with suppression of all glandular action. When death occurs it results from asphyxia or from slow failure of respiration and the circulation.

Diagnosis.—It is of great importance, therapeutically, to distinguish at once between attacks of sunstroke and heat exhaustion. This may be readily done by the aid of a thermometer. Cases of sunstroke are to be differentiated from cerebral hemorrhage and alcoholic insensibility by the history, season, occupation, and by the temperature.

Prognosis.—Attacks of heat exhaustion, if properly and promptly treated, are favorable. The prognosis of sunstroke, or heat-fever, is unfavorable in the majority of cases, death resulting in from half an hour to several hours. Unfavorable indications are, increased temperature, cardiac failure, convulsions, and absent reflexes, followed by complete muscular relaxation.

Favorable indications are decline in surface heat and axillary or rectal temperature, stronger pulse, increased depth of respirations, restored reflexes, and return of consciousness.

Sequels.—In any form of this affection one or more of the following conditions may result: headache, vertigo, insomnia, epilepsy, mental enfeeblement, and monoplegia, paraplegia, or hemiplegia.

Treatment.—In *heat exhaustion,* the patient should be placed in the recumbent posture with the head low and stimulants administered. Hot applications are of great value. If the patient is able to swallow, brandy, ℥ss-i (15-30 Cc.), with deodorized tincture of opium, ♏xv-xxx (1-2 Cc.), should be given at once and repeated if the occasion requires it. Aromatic spirits of ammonia, fʒi (4 Cc.), in hot water or milk every half hour is a useful adjunct. If the patient is unable to swallow, these remedies may be given by enema, or whiskey, strychnin sulphate, and tincture of digitalis may be used hypodermically. As convalescence begins, tonic doses of quinin hydrochlorate and strychnin sulphate should be prescribed.

In *sunstroke*, the indications for treatment are directly opposite. The patient is in imminent danger from the extraordinary temperature, and measures to reduce it must at once be instituted. Of these none give such excellent results as rubbing with ice, the cold bath or cold pack, and cold effusions, cold enemata, and the hypodermic use of quinin sulphate or antipyrin. The tendency to subsequent rise of temperature is met by wrapping the patient in a wet sheet and repeating the hypodermic medication, unless consciousness has been regained, when the remedies may be given by the mouth. If convulsions and restlessness occur, morphin sulphate, gr. ¼-½ (0.016-0.032 Gm.), hypodermically, cautiously repeated if necessary, or chloral or bromids by the rectum will be of value. In the occurrence of depression, strychnin sulphate, gr. 1-24 (0.0025 Gm.), repeated every half hour hypodermically, together with other modes of stimulation is indicated. Hypodermoclysis and enteroclysis may also be of value under such circumstances. During convalesence iron, quinin, and other tonics are required.

ACUTE HYDROCEPHALUS.

Synonyms.—Acquired hydrocephalus; serous apoplexy.

Definition.—Strictly speaking, hydrocephalus signifies water in the brain, but it is here restricted to the presence of a serous fluid in the arachnoid spaces, in the pia mater, in the ventricles, and in the brain substance (edema); characterized by the more or less sudden development of cerebral excitation, followed by depression and usually death.

Causes.—The affection is most common between the ages of one and five although it may occur at any age. A neurotic temperament is a rather strong predisposing factor. Among the exciting causes are unfavorable hygienic conditions, dentition, eruptive fevers, blows on the head, mechanical causes preventing the return of the blood from the veins of Galen and the right sinus, compression of the jugular vein, acute leptomeningitis, diseases of the right heart, and Bright's disease.

Pathologic Anatomy.—The effusion may be limited to the ventricles, although there is usually considerable distention of the subarachnoid spaces and edema of the pia mater and neighboring portions of the brain, whence results more or less softening, especially around the ventricles. The choroid plexus is hyperemic and may be the seat of minute extravasations.

Symptoms.—According to the grouping of the principal symptoms, acute hydrocephalus may be considered as of three varieties, *simple, convulsive,* and *comatose.*

Simple acute hydrocephalus is most common in children, and begins with feverishness, headache, vertigo, photophobia, restlessness, nocturnal delirium, insomnia, twitching, spasmodic contractions of the muscles, and great hyperesthesia of the skin. Such symptoms continue for several days, when convulsions occur, followed by death, or a continuance of the symptoms, followed by rigidity, stupor, and death.

Convulsive variety, usually the result of Bright's disease or a general dropsy, is ushered in with headache, nausea, and vomiting, followed in a day or two by convulsions, passing into coma, which usually terminates fatally, although rarely a remission may precede death for a day or two.

Comatose variety, known also as "serous apoplexy," begins abruptly with the phenomena of apoplexy, the result of the sudden effusion. The pressure is usually so great on the medulla oblongata that it ceases to functionate, death resulting usually in a few hours, rarely several days.

Prognosis.—Unfavorable.

Treatment.—The underlying disease should receive careful attention and the symptoms should be relieved as they arise. An attempt may be made to remove the fluid by diuretics and full doses of potassium iodid.

CONGENITAL HYDROCEPHALUS.

Synonym.—Chronic hydrocephalus.

Definition.—An excessive accumulation of the cerebrospinal fluid — a cerebral dropsy — in the ventricles — *internal hydrocephalus,* or in the meshes of the pia-arachnoid — *external hydrocephalus,* or in both — *mixed hydrocephalus;* characterized by enlargement of the head and more or less pronounced nervous phenomena.

Causes.—It is a disease of infants and young children, and is developed in the prenatal period. The affection occurs usually in the offspring of tubercular, scrofulous, or syphilitic parents. It may arise from imperfect or arrested development of the brain or its membranes and from inflammatory changes in the ventricles and ependyma. Occlusion of the passages by which the ventricles, and ventricles and subarachnoid space communicate is a cause in many cases.

Pathologic Anatomy.—Enlargement of the head is the chief external manifestation, but there is no constant ratio between the size of the head and the amount of fluid, the quantity varying from an ounce to a pint or more. The liquid is transparent, of a straw color, containing a small amount of albumin and chlorid of sodium. If the quantity of fluid be small, the ventricles are simply distended; if the amount be large, the optic thalami and corpus striatum are depressed and flattened, the roof of the ventricles thinned, and the foramen of Monro is greatly enlarged. The enlargement of the head may occur before birth and impede or prevent natural delivery, or the head may be normal at birth and increase afterward. As enlargement progresses, the bones are so thinned as to be translucent; the fontanelles and sutures are widened; the lateral portions of the cranium project; the forehead bulges out over the eyes; and the orbital plates are depressed, forcing the eyes outward and downward, producing a variety of exophthalmos; and the head has an irregular, triangular shape, the base of the triangle being the top of the head. The scalp being stretched by the pressure within, becomes tense and thin, and scantily covered with hair; the veins, which ramify in it, are unusually prominent and large, and the entire head is elastic on pressure, from the amount of liquid beneath.

In almost every case of internal hydrocephalus examined after death by Hilton, the cerebrospinal opening between the fourth ventricle and the spinal canal was found to be so completely closed that no cerebrospinal fluid could escape from the interior of the brain. From this, Hilton reasoned that the accumulation of fluid constituting this disease was due entirely to this obstruction.

Symptoms.—The first manifestation of the disease to attract attention is the increased size of the head in an emaciated child whose appetite is good and who seemingly partakes of food well. The head appears too heavy; the eyes are prominent and have a downward direction; the face is devoid of expression, old and wrinkled, the voice feeble; and the mental development is not in keeping with the age. When the period for standing or walking arrives, the power is found wanting. The further history is but a continuation and exaggeration of this state, until convulsions occur, which sooner or later terminate fatally. The course of congenital hydrocephalus is usually slow, but becomes progressively worse. The majority terminate within the first year; cases are recorded, however, of ten and fifteen years' duration.

Diagnosis.—In rachitis the volume of the head is increased, due, in part, at least, to a deposit of calcareous matter on the exterior of the cranial bones. Rachitis may be mistaken for hydrocephalus in cases in which the amount of liquid is small. The differential diagnosis is based on the shape of the head—round in rachitis, square or triangular or with prominences in hydrocephalus, with the persistent downward direction of the eyes and the elasticity of the head on pressure.

Prognosis.—Unfavorable. Arrest of progress and even cures have been reported. Spontaneous cures have been reported following the accidental discharge of the fluid, but such reports are exceptional.

Treatment.—The use of the finest aspirator needle to evacuate the fluid is fully justifiable. The proper situation for the puncture is the coronal suture, about an inch or an inch and a half from the anterior fontanelle. Firm but gentle compression of the cranium with adhesive strips should be made during the escape of the fluid and afterward. A few ounces of fluid only should be withdrawn at a time. The internal use of potassium iodid is recommended. All measures which tend to promote constructive metamorphosis are to be employed.

DISEASES OF THE SPINAL CORD.

SPINAL HYPEREMIA.

Synonyms.—Spinal congestion; plethora spinalis.

Definition.—An abnormal fullness of the vessels of the meninges and cord; *active* when an arterial hyperemia; *passive* when a venous hyperemia; characterized by pain in the back, with more or less pronounced disorders of sensation and locomotion.

Causes.—Cold and exposure; arrested menses; arrest of a habitual hemorrhoidal discharge; malaria; protracted erect posture; injuries to the back; certain spinal poisons, as strychnin, picrotoxin, and alcoholic excesses.

Pathologic Anatomy.—*Active.* The post-mortem appearances are congestion of the meninges and cord, the same vessels supplying both, with numerous points of extravasation, due to the rupture of capillary vessels. The spinal fluid is increased in amount.

Passive. A general bluish discoloration, owing to the abnormal fullness of the large anastomosing vessels; the spinal fluid is somewhat increased.

Symptoms.—*Active hyperemia* is manifested by dull pain in the dorsal or lumbar region, shooting into the hips and thighs, persistent and increased by pressure; tenderness on motion; tingling sensations in the limbs and feet, and sometimes in the hands and arms; a feeling of constriction about the abdomen is often present, with rigidity of the abdominal muscles; increased reflexes, with disorders of motility, and when the patient is in the recumbent position, jerking of the limbs. Walking is accomplished with difficulty, from an incomplete loss of power. If the upper part of the cord be affected, dyspnea and palpitation will occur.

There may also occur painful priapism and frequent nocturnal emissions.

The above symptoms may be followed by a more or less pronounced temporary depression, the sensation diminished, and the lower limbs benumbed and heavy, the movements being weak. The electro-contractility is preserved, and in many cases even increased or exaggerated.

Duration.—The affection lasts from a few hours to several days, and when unduly prolonged terminates in myelitis.

Diagnosis.—Anemia causes more or less spinal irritability and tenderness; but the history, pallor, and general weakness, unassociated with defects of motility or sensibility, will prevent error.

Spinal meningeal hemorrhage is more sudden in its onset, is more violent, and has a greater range of symptoms than spinal hyperemia.

Myelitis and spinal meningitis have symptoms in common with spinal congestion, which will be pointed out when discussing those conditions.

Prognosis.—The outlook is favorable, recovery usually taking place in three or four days. If the symptoms show a tendency to linger, myelitis, more or less pronounced, will ensue.

Treatment.—Rest is indicated, but the patient should avoid lying on the back. Cups or leeches should be applied along the spine followed either by the iced or the hot douche or hot sponges. Active purgation should be brought about to lessen the blood-pressure. When the condition is due to arrested perspiration a hot-air bath and the administration of pilocarpin are of value. When it follows arrest of the menses, aconite will be of benefit. If associated with a very active circulation, potassium bromid, fluid extract of gelsemium, ♏v (0.3 Cc.), or fluid extract of ergot, fʒss-i (2-4 Cc.), will afford great relief.

In passive hyperemia, the cause should be ascertained and removed. Ergot, digitalis, tonics, and purgatives will serve to lessen the congestion.

SPINAL PACHYMENINGITIS.

Synonyms.—Pachymeningitis spinalis interna; hypertrophic pachymeningitis; pseudo-membranous pachymeningitis.

Definition.—An inflammation of the inner surface of the spinal dura mater characterized by an exudation upon this inner surface, attended by violent pains in the head, neck, shoulders and arms, followed by muscular contractures and paralyses of the upper extremities.

Causes.—Exposure to cold and damp, alcoholism, syphilis, gout, and injuries are the most common causes. It may be secondary to Pott's disease.

Pathologic Anatomy.—*Hypertrophic pachymeningitis* is characterized by an exudation upon the inner surface of the spinal dura mater, which gradually solidifies into a layer of compact connective-tissue. This membrane presses upon the spinal cord and nerves producing myelitis and optic neuritis with subsequent muscular atrophy. The most frequent seat of this form of the affection is the cervical region and it is then termed *cervical hypertrophic pachymeningitis.*

In the *pseudomembranous variety,* an exudation also forms in which large numbers of blood-vessels develop and rupture, the resulting extravasation forming a cyst or hematoma which exerts considerable pressure on the cord and nerves.

Symptoms.—The onset is slow and gradual, with irregular chills and feverishness, more or less continuous violent pains with stiffness in the head, neck, shoulders, and arms, and a painful sense of constriction of the upper thorax. Numbness and pricking in the arms are often present. Occasionally nausea and vomiting occur. These symptoms may continue in varying degrees of severity for several months, the muscles of the painful parts ultimately undergoing atrophy, followed by spasmodic contraction, particularly of the hands and wrists, and eventually by paralysis. The paralytic stage develops gradually with weakness in the arms associated with contractures and rigidity. The pain continues and there may be anesthesia, hyperesthesia, or paresthesia. Trophic changes are common. Later, there develop paraplegia with rigidity, exaggerated reflexes and spinal epilepsy. The electro-contractility is lost. It has been observed, clinically, that the immediate cause of death in chronic cerebral and spinal disease is to be found in an intercurrent attack of nephritis or tuberculosis.

Prognosis.—If early recognized and promptly treated, the hypertrophic form may be improved. Generally, however, the prognosis is unfavorable.

Treatment.—Rest in bed with repeated counterirritaion over the spine is indicated. The diet should be highly nutritious and drugs such as cod-liver oil, hypophosphites, and potassium iodid should be administered. The various symptoms, such as pains and spasms, should be treated as they arise on general principles.

SPINAL MENINGITIS.

Synonym.—Leptomeningitis spinalis.

Definition.—Inflammation of the arachnoid and pia mater membranes of the spinal cord, either acute, subacute, or chronic; characterized by pain in the back, rigidity of the muscles, and disorders of motility and sensibility.

Causes.—The disease is rare and is nearly always due to an infection. Cerebrospinal meningitis, tuberculosis, syphilis, typhoid fever, septicemia, traumatism, and exposure are the most common causes.

Pathologic Anatomy.—The *acute form* is attended by hyperemia of the membranes with swelling of the tissues, the result of serous infiltration, followed by purulent and fibrinous exudations. The roots of the spinal nerves are covered with exudation, and are swollen and soft. The cord proper is more or less congested and edematous.

In the *chronic form,* there is adhesion of the membranes, with more or less accumulation of fluid, resulting in atrophic degeneration of the cord from pressure. When the disease is tuberculous in origin, granulations are seen distributed over the pia, arachnoid, and inner surface of the dura.

Symptoms.—There are two stages: the first, the stage of *irritation;* the second, the stage of *paralysis* of motion and sensation, with atrophy. Although an inflammatory affection, its onset is usually subacute, the febrile reaction being moderate, with intense boring pain in the back, aggravated by motion, rigidity of the spine, and a sense of constriction around the body,—"the girdle." Spasmodic contractions of the muscles supplied by the nerves originating at the seat of the lesion, with inability to straighten the limbs are also present. If the lower part of the spinal membranes is the seat, there occur retention of urine and constipation; if upper part, dysphagia, dyspnea, and feeble heart. If the inflammation extend to the medulla, the above symptoms are associated with disorders of speech, vomiting, and delirium. The muscular contractions are excited or increased by motion, but uninfluenced by pressure. Reflex movements are not abolished. The rigidity and spasmodic contractions of the muscles are followed by paralysis, more or less complete, death ensuing from paralysis of the muscles of respiration.

Electro-contractility is lessened or absent, both as to motility and sensibility in the affected parts.

The *chronic form* succeeds to the acute or originates spontaneously, and presents the same form and order of symptoms—excitation or irritation, and depression or paralysis.

Diagnosis.—The points of importance are: deep, boring pain in the back, aggravated by motion but not by pressure, with spasmodic contraction of the muscles, followed by paralysis.

Myelitis is marked by slight, or no pain, with earlier and more complete paralysis.

Tetanus may be confounded with spinal meningitis. The points of distinction are: in the former occur early trismus with rhythmic spasms excited by irritation of the skin, whereas irritation of the skin does not, in spinal meningitis, produce muscular contractions, but movement of the limbs does; tetanus progressively increases, and is not associated with fever; there is usually a clear history of an injury.

Prognosis.—Generally unfavorable. Death is either sudden, from paralysis of respiration and of the heart, or gradually, the result of exhaustion.

Critical discharges, such as profuse perspiration, urinary flow, or epistaxis occasionally occur, and are followed by rapid recovery. Cases recovering may have more or less pronounced partial or complete paralysis.

Treatment.—The patient should be placed at rest in bed and allowed to lie on the side or face. Cups or leeches should be applied along the spine, followed by ice, the hot douche, hot sponges, or mustard. Free purgation should be obtained. In cases due to syphilis, mercury and the iodids should be given in full doses. In the paralytic stage, quinin sulphate, gr. iii (0.2 Gm.), with alcoholic extract of belladonna, gr. ¼ (0.016 Gm.), three times daily, is often of great value. The galvanic current should be applied to the spine and to the nerve trunks and the faradic current to the affected muscles. Deep injections of strychnin and massage should also be employed.

ACUTE MYELITIS.

Synonyms.—Acute or general diffuse myelitis; transverse myelitis; softening of the cord.

Definition.—An inflammation affecting the substance of the spinal cord, which may be limited to the gray or white matter, and may involve the whole or isolated portions of the cord. When the gray matter alone is inflamed, it is termed *central myelitis;* when the white matter and the meninges, it is termed *cortical myelitis;* it may be ascending, descending, or transverse in its extension. The disease is characterized by more or less sudden and complete loss of motion and sensation.

Causes.—It may follow acute congestion or spinal meningitis, or it may be due to exposure to cold and damp or wet weather, injuries to the vertebræ, syphilis, rheumatism, puerperal fever, typhus fever, exanthemata, or to poisoning by lead, arsenic, or mercury.

Pathologic Anatomy.—The substance of the cord is intensely hyperemic and extravasations are scattered throughout it giving to the tissues a reddish-brown or chocolate tint. Sometimes prominent hemorrhagic effusions are observed. Serous transudations are also present resulting in softening of the structure of the cord, the color changing to yellow and white; the nerve-elements undergo fatty degeneration and present the appearance and consistency of cream. The membranes are involved in more or less similar changes. The microscope reveals degeneration of the cellular elements and their replacement by fat granules, granular débris, and blood-cells.

Symptoms.—The severity of the symptoms depends upon the extent and location of the inflammation.

The onset is usually sudden, with a chill, fever, 103° F., frequent pulse, and alterations in sensibility and motility — to wit:

pain in the back, aggravated by touch and by heat and cold, with sensations of formication ("pins and needles"), the limb feeling as if asleep, or complete anesthesia, associated with severe neuralgic pains. The sensation of constriction around the body and limbs, as if encircled by a tight cord, "the girdle pains," is a characteristic symptom, and is followed by a rapidly developing paraplegia which becomes complete in a few hours and is accompanied by involuntary discharges. The reflex functions are usually abolished, as seen by attempting to cause movement of the limbs by tickling the feet or by striking the patella tendon; rarely are they diminished, very rarely exaggerated. The temperature of the affected limbs is lowered three or four degrees. Sloughs and bed-sores, and muscular atrophy result if the anterior cornua—the trophic centers—are affected.

The symptoms of loss of motion and sensibility, with rectal and vesical paralysis, are associated with more or less pronounced vomiting, hepatic disorders, irregularity of the heart, dyspnea, dysphagia, apnea, and painful priapism. The urine is markedly alkaline in reaction, finally developing cystitis. Among the late manifestations are shooting pains and spasmodic twitchings or contractions of one or all of the muscles of the paralyzed parts. The electro-contractility is abolished in the paralyzed parts.

Diagnosis.—The principal diagnostic features of acute myelitis are the "girdle" around the limbs or body, rapid and complete paraplegia, loss of sensation, lowered temperature in the affected parts, early and persistent sloughing (bed-sores), and alkaline urine or cystitis.

The diagnosis of the *location of the lesion* is made by a study of the degree of the anesthesia, the skin reflexes, and the distribution and extent of the paralysis, which are shown in the following table from Dana:

LOCALIZATION OF THE FUNCTIONS OF THE SEGMENTS OF THE SPINAL CORD.

Segment.	Muscles.	Reflex and Centers.	Sensation.
First cervical.	Rectus laterales. Rectus capitis. Anticus and posticus. Sterno-hyoid. Sterno-thryoid.		
Second and third cervical.	Sterno-mastoid. Trapezius. Scaleni and neck. Omo-hyoid. Diaphragm.	*Hypochondrium* (?). Sudden inspiration produced by sudden pressure beneath the lower border of ribs.	Backof head to vertex and neck. (Occipitalis major, occipitalis minor, auricularis magnus, superficialis colli, and supraclavicular.)
Fourth cervical.	Diaphragm. Deltoid. Biceps. Coraco-brachialis. Supinator-longus. Rhomboid. Supra- and infra-spinatus.	*Pupillary* (fourth cervical to second dorsal). Dilatation of the pupil produced by irritation of neck.	Neck. Shoulder, anterior surface. Outer arm. (Supraclavicular, circumflex, external musculo-cutaneous, cutaneous.)
Fifth cervical.	Deltoid. Biceps. Coraco-brachialis. Brachialis anticus. Supinator-longus. Supinator-brevis. Deep muscles of shoulder-blade. Rhomboid. Teres minor. Pectoralis (clavicular part).	*Scapular* (fifth cervical to first dorsal). Irritation of skin over the scapula produces contraction of scapular muscles. *Supinator longus.* Tapping the tendon of the supinator longus produces flexion of forearm.	Back of shoulder and arm. Outer side of arm and forearm to the wrist. (Supraclavicular, circumflex, external cutaneous, internal cutaneous, posterior spinal branches.)
Sixth cervical.	Serratus magnus. Deltoid. Biceps. Brachialis anticus. Subscapular. Pectoralis (clavicular part). Serratus magnus. Triceps. Pronators. Rhomboid. Latissimus dorsi.	*Triceps* (fifth to sixth cervical). Tapping elbow tendon produces extension of forearm. *Posterior wrist* (sixth to eighth cervical). Tapping tendons causes extension of hand.	Outer side and front of forearm. Back of hand, radial distribution. (Chiefly external cutaneous, internal cutaneous, radial.)
Seventh cervical.	Triceps (long head). Extensors of wrist and fingers. Pronators of wrist. Flexors of wrist. Subscapular. Pectoralis (costal) Latissimus dorsi. Teres major.	*Anterior wrist* (seventh to eighth cervical). Tapping anterior tendons causes flexion of wrist. *Palmar* (seventh cervical to first dorsal). Stroking palm causes closure of fingers.	Radial distribution in the hand. Median distribution in the palm, thumb, index, and one-half middle finger. (External cutaneous, internal cutaneous, radial, median, posterior spinal branches.)
Eighth cervical.	Triceps (long head). Flexors of wrist and fingers. Intrinsic hand muscles.	....	Ulnar area of hand, back and palm, inner border of forearm. (Internal cutaneous ulnar.)

LOCALIZATION OF THE FUNCTIONS OF THE SEGMENTS OF THE SPINAL CORD.—*Continued.*

SEGMENT.	MUSCLES.	REFLEX AND CENTERS.	SENSATION.
First dorsal.	Extensors of thumb. Intrinsic hand muscles. Thenar and hypothenar muscles.	....	Chiefly inner side of forearm and arm to near the axilla. (Chiefly internal cutaneous and nerve of Wrisberg or lesser internal cutaneous).
Second dorsal.	....	....	Inner side of arm near and in axilla. (Intercosto-humeral.)
Second to twelfth dorsal.	Muscles of back and abdomen. Erectores spinæ.	*Epigastric* (fourth to seventh dorsal). Tickling mammary region causes retraction of the epigastrium. *Abdominal* (seventh to eleventh dorsal). Stroking side of abdomen causes retraction of belly. Vasomotor centers. Second dorsal to second lumbar.	Skin of chest and abdomen, in bands running around and downward corresponding to spinal nerves. Upper gluteal region. (Intercostals and dorsal posterior nerves.)
First lumbar.	None.	*Cremasteric* (first to third lumbar). Stroking inner thigh causes retraction of scrotum.	Skin over groin and front of scrotum. (Ilio-hypogastric ilio-inguinal.)
Second lumbar.	Vastus internus.	*Patellar.* Striking patellar tendon causes extension of leg.	Outer side and upper front of thigh. Lumbar region. (Genito-crural, external cutaneous).
Third lumbar.	Sartorius; adductors of thigh.	....	Front and outer side of thigh. Inner side of leg and foot.
Fourth lumbar.	Flexors of thigh. Extensors of knee. Abductors of thigh.	*Gluteal* (fourth to fifth lumbar). Stroking buttock causes dimpling in fold of buttock.	Inner side of thigh, leg,and foot. (Internal cutaneous, long saphenous, obturator).
Fifth lumbar.	Outward rotators. Flexors of knee. Flexors of ankle. Peronei. Extensors of toes.	*Achilles tendon.* Over-extension causes rapid flexion of ankle, called ankle clonus.	Back of thigh and outer side of leg and ankle; sole; dorsum of foot. (External popliteal, external saphenous, musculo-cutaneous, plantar).
First and second sacral.	Calf muscles. Glutei. Peronei. Extensors of ankle. Small muscles of foot.	*Plantar* (fifth lumbar to second sacral). Tickling sole of foot causes flexion of toes and retraction of leg.	Back of buttock and thigh; side of leg and ankle; sole; dorsum of foot.
Third, fourth, and fifth sacral.	Peronei. Muscles of bladder, rectum, and external genitals.	Genital center. Vesical center. Anal center.	Circumanal region, anus, rectum, penis, urethra, vagina, perineum. (Small sciatic, pudic, inferior hemorrhoidal, inferior pudenal).

Acute spinal meningitis is distinguished from acute myelitis by severe pains, increased by pressure, with muscular contractions increased by motion, followed by paralysis much less profound than the paraplegia of myelitis; in spinal meningitis there exists cutaneous and muscular hyperesthesia, which are absent in myelitis.

Congestion of the spinal cord is characterized by the mild character and short duration of all the symptoms.

Hemorrhage in the spinal cord is abrupt with irritative symptoms, slight paralysis, preserved reflexes, and electro-contractility.

Hysteric paraplegia shows no trophic changes, no altered reflexes, slight atrophy, irregular anesthesia, contractures with impaired sensation of the contracted limb, and the presence of the stigmata of hysteria.

Lithemic paresthesia, characterized by tingling and numbness of fingers and toes, might lead to error if the cerebral symptoms of lithemia are overlooked.

Prognosis.—This depends upon the location of the lesion and completeness of the symptoms. If the paralysis is of the ascending variety, death occurs within a few days, from paralysis of the muscles of respiration. If the trophic centers are affected, there occur bed-sores, intense pyelo-nephritis and cystitis, and changes in the joints; death results from exhaustion in several weeks. Central myelitis, or inflammation of the gray matter, is rapid in its progress, death occurring in a week or two. The morbid process may in rare instances be arrested and the general health restored, but some spinal symptoms will persist.

Treatment.—Absolute rest is essential to even secure a palliation of the symptoms.

Locally, considerable relief follows the use of hot-water bags or sponges dipped in hot water and applied along the spine every few hours.

Internally, digitalis, strychnin sulphate, ergot, belladonna, bromids, iodids, cimicifuga, quinin sulphate, and other similar drugs may be employed but the results following their use is somewhat doubtful. Careful nursing is, however, of great benefit. Absolute cleanliness and frequent change of posture is necessary to prevent bed-sores. Retention of urine should be avoided by frequent aseptic catheterization. Cystitis should be treated by boric acid irrigations. The condition of the intestinal tract should also receive attention. If the affection shows any tendency towards recovery, this tendency should be stimulated by electricity and massage.

INFANTILE SPINAL PARALYSIS.

Synonyms.—Myelitis of the anterior horns; poliomyelitis anterior acuta; essential paralysis of children; atrophic paralysis of children.

Definition.—A rapidly developed inflammation of the anterior horns of the gray matter of the cord, occurring suddenly in children, occasionally in adults,—acute spinal paralysis of adults,—characterized by mild fever, muscular tremors and twitchings, and paralysis of groups of muscles, followed by more or less atrophy.

Causes.—It is essentially a disease of early life, from the second month to the third or fourth year, but it may rarely be observed in adults. The affection is more common during the summer months, and males are most often attacked. It occasionally appears in epidemic form. The real cause is unknown. Exposure to cold and damp, dentition, injuries, and the infectious fevers may act as predisposing causes.

Pathologic Anatomy.—The early changes are: Medullary hyperemia, vascular exudation, and inflammatory softening, although the naked eye may not recognize any changes. Microscopic examination reveals inflammatory softening of the anterior horns of the gray matter. Among other constant lesions are atrophic degeneration of the multipolar ganglion-cells and of the anterior nerve-roots. The changes noted as occurring in the cord are usually limited to the dorso-lumbar and cervical enlargements.

As a direct result of the changes in the trophic centers and the nerve degeneration of the muscular fibers supplied, there ensue changes in the bones and joints, leading to great deformities.

Symptoms.—The onset of the affection varies; it may be acute, subacute, or chronic; it is usually sudden, with an attack of mild fever of a remittent type, of a few days' duration, on recovery from which it is noticed that the child is paralyzed. Rarely, the paralysis may be preceded by convulsions.

The paralysis may affect both arms and both legs, the legs alone, or only one of the four extremities; it may, very rarely, be a hemiplegia. As a rule, however, the leg suffers more frequently than the arm: in paralysis of the leg the muscles below the knee suffer more severely than those above. The bladder and rectum are not affected, or if so, only temporarily, and anesthesia or numbness cannot be detected. The temperature of the paralyzed limb is low and the part is cyanosed in appearance. After a few days there is a slight improvement in the paralyzed parts, although the muscles show a rapid wasting, which is progressive until all muscular tissue is gone. The reflex movements are impaired or abolished.

The electro-contractility by the faradic current is abolished in the paralyzed parts.

With the galvanic or constant current the "reactions of degeneration" are developed. To fully understand the meaning of this term a knowledge of the normal electrical reactions is necessary.

The normal formulas for the production of muscular contraction in the physiologic state are as follows, the strength of the current being barely capable of causing fair contractions:

First. The most effective contractions are produced by the cathode (negative) pole on closing the circuit (C. C.).

Second. The second most effective are produced by the anode (positive) pole on closing the circuit (A. C.).

Third. The next most effective is by the anode pole on opening the circuit (A. O.).

Fourth. Cathode pole contractions on opening circuit are rarely seen in the physiologic state (C. O.).

The "reactions of degeneration" are shown by any reversal of the regular formulas—to wit: if the anodal closure (A. C.); shows stronger contractions than cathodal closure (C. C.); still greater degeneration is shown if anodal opening (A. O.) contractions are stronger than either of the above; and most complete degeneration is shown by the complete reversal of the normal formulas as shown by distinct cathodal opening (C. O.) contractions.

Sequels.—Among the deformities resulting from the paralysis are the different forms of talipes.

Talipes equinus, the result of paralysis of the antero-external muscular group of the leg.

Equino-varus, the result of paralysis of the antero-external muscular group of the leg, together with the adductors of the foot.

Talipes calcaneus, the result of paralysis of the muscles of the calf of the leg.

Talipes cavus,—"pes cavus,"—characterized by the hollowing of the sole of the foot, with prominence of the instep, the result of paralysis of the calf muscles with contraction of the long flexor of the toe or the long peroneus—the foot flexors.

Diagnosis.—The recognition of acute poliomyelitis is not always possible at the onset or during the early days of its course, as localized paralyses are difficult of detection in children, but *immobility of one leg or arm* in children with febrile symptoms, or following convulsions is always an indication of poliomyelitis. After the initial stage has passed, the presence of paralysis, wasting, presence of R. D. (reactions of degeneration), loss of reflexes, and the absence of anesthesia, render the diagnosis very easy.

Hemiplegia from acute cerebral affections in children can be distinguished from infantile paralysis by the disorders of intelligence and the special senses, and the perseverance of the normal electro-contractility.

Paralysis of myelitis occurs in older persons, and is associated with disturbances of the genitourinary organs and bed-sores.

Pseudo-muscular hypertrophy, with paralysis, begins gradually, becoming progressively worse with increase in the size of the limbs.

Prognosis.—Except in cases in which the onset is very severe, the outlook as regards life is good. More or less paralysis with muscular wasting and deformities always results, but by its early recognition and prompt treatment the extent may be greatly lessened.

Treatment.—During the febrile stage the patient should be placed at rest in bed and all the secretions rendered free. If the affection is suspected at this period, the limbs should be wrapped in cotton-wool and ergot administered to lessen the spinal congestion. Counterirritation is unnecessary. As soon as the febrile reaction has subsided and the paralysis becomes manifest the child should be well fed and taken out-doors once daily. Gentle friction should be applied to the affected muscles at first,, followed later by the hot spinal douche and mild galvanism. Internally, quinin, belladonna, ergot, and potassium iodid may be of value. Later, as improvement takes place tincture of nux vomica, ♏ i-iii (0.06-0.2 Gm.), three times daily, or hypodermic injections of strychnin sulphate, gr. 1-16 - 1-100 (0.004-0.00065 Gm.), according to the age, twice a week, and faradism to the paralyzed muscles are to be used. Means should be taken to prevent deformities.

CHRONIC PROGRESSIVE BULBAR PARALYSIS.

Synonyms.—Glosso-labio-laryngeal paralysis; bulbar paralysis.

Definition.—A chronic degenerative affection of certain nuclei of the medulla oblongata, characterized by a slowly progressive bilateral paralysis of the tongue, lips, palate, pharynx, and larynx, with atrophy of the tongue and lips.

Causes.—The etiology is obscure. It rarely occurs before the fortieth year. It may be brought about by extension of spinal or cerebral affections to the medulla. Among other etiologic influences may be mentioned cold, rheumatism, gout, syphilis, and injuries about the neck.

Pathologic Anatomy.—The structural changes consist in degenerative atrophy of the gray nuclei in the floor of the fourth ventricle, with atrophy and gray discoloration of the nerve-roots from the medulla, especially of the facial and hypoglossal nerves. The motor ganglion-cells atrophy and disappear, not infrequently being the only changes. The nerves supplied to the muscles exhibit sclerosis of the neurilemma, and degenerative atrophy is found in the nerve-roots coming from the bulb.

Symptoms.—The disease begins insidiously. There is first noticed some difficulty in articulation, from want of precision in movements of the tongue, particularly in the use of the lingual consonants, *l*, *n*, *r*, and *t*, which increases until that organ is completely paralyzed. The paralysis gradually invades the soft palate and pharyngeal muscles, causing difficulty in deglutition; the orbicularis oris preventing closure of the lips; the laryngeal muscles, interfering with articulation. With the increasing loss of power in the tongue and lips there is also a gradual atrophy of these muscles; the atrophy usually antedates the paralysis. When the disease is fully developed, the condition of the patient is most pitiable; articulation is impaired or impossible, and deglutition interfered with, the lips remaining apart allowing the saliva to dribble from the mouth and liquids to return through the nose with attempts at swallowing. As the malady progresses, the pneumogastric nucleus becomes involved, resulting in loss of voice, difficulty of respiration, and cardiac irregularity. The general health gradually suffers from insufficient nutrition and imperfect respiration, although the mind is clear until the end. The "reactions of degeneration" are present.

Besides the chronic bulbar paralysis, there are two acute forms with the same symptoms as the chronic cases, only they develop suddenly, one, the result of hemorrhage into the medulla, which at the onset has vertigo, vomiting, loss of power in the limbs, and slight sensory disturbances, all of which disappear, leaving the glosso-labio-laryngeal paralysis; the second form comes suddenly, with fever, vomiting, and loss of power in the limbs, soon disappearing, leaving the characteristic bulbar symptoms; this variety is inflammatory and closely allied to acute poliomyelitis.

Diagnosis.—The recognition of this disease is not difficult. The paralysis of deglutition is particularly characteristic.

Prognosis.—The acute forms terminate fatally within a few days. The chronic form lasts from one to five years and ultimately terminates in death from exhaustion, respiratory failure, or cardiac failure.

Treatment.—The treatment is entirely symptomatic. Feeding should be accomplished by the stomach-tube to avoid pulmonary aspiration. Massage and galvanism should be employed.

PROGRESSIVE MUSCULAR ATROPHY.

Synonyms.—Wasting palsy; chronic spinal muscular atrophy; chronic poliomyelitis; amyotrophic lateral sclerosis.

Definition.—A chronic progressive motor paralysis with atrophy of certain groups of muscles. The paralysis proportionate to the wasting or fibrillary atrophy.

Causes.—It occurs most frequently in males between twenty-five and fifty years of age and in many instances is hereditary. A predisposition seems to exist in those who habitually use one set of muscles (muscular strain). Exposure to cold and damp. lead; syphilis; injuries to the spinal column and acute diseases as diphtheria, measles, acute rheumatism, typhoid and typhus fevers, may influence its production.

Pathologic Anatcmy.—Two theories as to the origin of the pathologic changes are held: one that the initial lesion is in the cord (Charcot), the other in the musclar interstitial connective-tissue (Friedreich).

The morbid alterations are of two groups—spinal and muscular.

The spinal changes consist in the atrophy and degeneration of the anterior columns, wasting and disappearance of the multipolar ganglion-cells of the anterior horns with hyperplasia of the neuroglia; rarely, the hyperplasia extends to the lateral columns (amyotrophic lateral sclerosis); also atrophy, and degeneration of the anterior nerve-roots.

The muscular changes consist of a progressive wasting of the muscular tissue, with increase of the interstitial connective-tissue. "The final result is that the muscle is converted into a mere fibrous band with numerous fat-cells, the development of this latter material taking place outside of the muscular elements and in the newly-formed connective-tissue" (Bartholow).

Symptoms.—The invasion is gradual, the disease having been in progress some weeks or months before the patient is aware of its existence.

In the immense majority of cases, the disease is permanently limited to one or a few groups of muscles in the upper, or more rarely in the lower, extremities. The only muscles not yet known to be attacked are those of mastication and those that move the eyeball (Roberts).

Fibrillary contraction is an early symptom, continuing more or less marked so long as any muscular fibers remain. It consists of wave-like movements of the muscles, excited automatically, by draughts of air or percussion. Coincident with the wasting there occur loss of power, disorders of sensation, and coolness and pallor of the surface.

The natural roundness and contour of the body and limbs are changed, the bones standing out with unusual distinctness, giving the individual the appearance of a skeleton clothed in skin.

Four types of the disease are recognized: 1, the hand-type; 2, the juvenile type; 3, the infantile facial type; 4, the peroneal type.

The *hand-type:* wasting begins in the hand, particularly in the short muscles of the thumb and the ball of the little finger—the thenar and hypothenar eminences. The complete atrophy of the thumb muscles produces such a change in the shape of the hand as to give it the name of the *ape-hand.* Soon, and may be at the same time, wasting of the dorsal interosseous muscles is observed, with consequent loss of power in these muscles, producing the deformity known as *claw-hand.* Shortly the deltoid and other arm muscles are involved in the wasting and contraction.

The *juvenile type* (Erb): a rare form, affecting the muscles of the shoulder and upper arm, and less commonly the muscles of the lower extremities. This form follows the hand-type after a time, but Erb described cases occurring primarily in these parts. Rarely, wasting in the suprascapular muscles, with fibrillary contractions, is seen alone.

The *infantile facial type* involves the muscles of expression, changing entirely the appearance of the individual and giving the eyeballs undue prominence from atrophy of the surrounding muscles, not unlike exophthalmos. After a time, the muscles of the shoulder and arm are involved, except the supraspinatis, infraspinatis, and the flexors of the hand and fingers.

The *peroneal type:* wasting first appears in the muscles of the legs, extending to the feet, producing single or double club-foot. After a time the muscles of the hands and arms are involved, with the consequent deformities. Vasomotor changes are observed in this type.

Rarely, all the types are more or less blended in the same individual. Usually, the electro-contractility is preserved so long as muscular fibers remain.

Diagnosis.—When wasting palsy is fully developed, its diagnosis is a simple matter. In its early stages a doubt may exist, but attention to the history, symptoms, and progress will determine the question.

Syringomyelia often begins with muscular atrophy as a marked symptom, and may be confounded with wasting palsy, the chief points of distinction between which are: the loss of power of perceiving heat, or, often, to distinguish between heat and cold, and the appearance of trophic changes, such as a dusky or purplish hue of the hands, with a uniform thickness resembling myxedema, the development of blebs and ulcers, changes in the nails, and sometimes arthropathies in the former.

Prognosis.—Very unfavorable, although the danger to life is often very remote. The disease may be arrested and remain stationary for years.

Treatment.—Internal medication seems to have little or no effect on the malady. In syphilitic cases, mercury and potassium iodid should be administered and if mineral poisoning is suspected potassium iodid, alone, should be given. A generous diet together with the administration of drugs such as arsenic, strychnin sulphate, and cod-liver oil is indicated in all cases. If the disease is the result of over-exertion of any group or groups of muscles, these groups should be placed at absolute rest. Galvanism applied locally to the affected muscles is of great benefit. Faradism is also of value. Massage, friction, and hot sponging are useful adjuvants to the treatment. The injection of glycerin solution into the wasting muscles in a case confined to the left upper extremity was followed by great improvement (Bartholow).

PSEUDO-HYPERTROPHIC MUSCULAR PARALYSIS.

Synonyms.—Pseudo-muscular hypertrophy; lipomatous muscular atrophy; lipomatosis luxurians muscularis.

Definition.—A diseased condition of the muscles in which the muscle-fibers undergo atrophy and are replaced by adipose and connective-tissue, causing weakness and enlargement of the muscles.

Causes.—The condition is first noticed in childhood and is markedly influenced by heredity. Certain families seem especially predisposed. Boys seem more liable to be affected than girls.

Pathology.—Except in very rare instances the structural changes are confined to the affected muscles. Microscopic examination reveals atrophy of the individual muscle-fibers with the disposition of adipose and connective tissue between them.

Symptoms.—Weakness of the muscles with awkwardness and clumsiness in performance of ordinary movements such as walking, is the earliest symptom. This progresses and paralysis of the extremities with muscular enlargement is soon manifest. On rising from a recumbent posture the patient gets up on all-fours, raising the trunk by moving the hands upon the floor, eventually becoming erect by pushing himself up by his hands on the knees. The gait is waddling. Electric excitability is diminished but the reactions of degeneration are never obtained. Reflexes are lessened and sometimes absent. Disturbances of sensation and mental disorders are absent. As the disease progresses, the patient ultimately becomes bed-ridden although apparently well-preserved for a long period. The course is indefinite and the outlook is unfavorable. Death usually results from some intercurrent affection. Treatment is of no avail.

ACUTE ASCENDING PARALYSIS.

Synonym.—Landry's Disease.

Definition.—An acute disease characterized by palsy beginning in the feet and ascending to other muscles of the body, finally involving the medulla. Pain and trophic disturbances are absent. The reflexes are diminished and the sphincters are not involved. The affection is rare and occurs most often in young male adults. The etiology and pathology are obscure. The onset is sudden and the course acute, terminating usually in death within a week, occasionally being prolonged three or four weeks. The treatment is unsatisfactory.

SPINAL SCLEROSIS.

Synonym.—Duchenne's disease.

Definition.—A myelitis; an increase in the connective-tissue of the spinal cord, with atrophy of the nerve-structure proper.

Varieties.—I. *Lateral sclerosis.* II. *Posterior sclerosis*, or *locomotor ataxia.* III. *Ataxic paraplegia.* IV. *Cerebrospinal sclerosis.*

Causes.—Generally there is an hereditary neuropathic predisposition. The affection occurs most often in males between the ages of thirty-five and fifty-five. Among the principal etiologic factors may be mentioned syphilis, alcoholism, mineral poisons, shock or injuries to the cord, over-exertion, and exposure to cold and wet. It is said that railroad enginemen and firemen, as well as conductors and other trainmen, suffer from this and other spinal diseases by reason of the continual concussion of railway travel. The freedom from the disease in the negro has been noted by Mitchell.

Pathologic Anatomy.—The changes in the cord are gradual in their development and follow a longitudinal instead of a transverse direction. The form, consistency, and color of the cord are altered, it being atrophied, indurated, and of a grayish color. The changes are hyperplasia of the connective-tissue, with granular degeneration, atrophy and disappearance of the nerve-elements proper. The nerve-roots undergo the same fibroid change. The joints undergo remarkable atrophic degeneration — the arthropathies or Charcot-joints, consisting of an osseous hyperplasia, the joint enlarging to an enormous extent.

PRIMARY LATERAL SCLEROSIS.

Synonyms.—Antero-lateral sclerosis; spasmodic tabes dorsalis (Charcot); spastic spinal paralysis (Erb); amyotrophic lateral sclerosis.

Definition.—A degeneration of the lateral columns of the cord, characterized by paraplegia, contractures of the muscles, with exaggerated reflexes.

Pathology.—The exact morbid condition is still a subject of discussion. The site of the lesion is the lateral white columns, in some cases extending to the anterior horn, and involving the whole length of the cord. The changes consist in an interstitial hyperplasia of the connective-tissue, and an atrophy of the nerve-elements.

Symptoms.—The onset of the disease is very gradual, with increasing feeling of heaviness and weakness in the limbs, progressing to a complete paraplegia. There are also jerking and twitching with cramps and stiffness of the muscles of the paretic limbs.

The spasms of the legs gradually increase in extent as the power lessens, until at last the legs, whenever extended, pass into a condition of strong extensor spasm, rigidly fixing them to the pelvis, so that the patient lies rigid; if one leg is lifted from the couch by the observer, the other leg is moved also. The spasm may be such that the knee cannot be passively flexed by any force that can be applied to it, until the spasm has lessened. When flexed, the limb is comparatively supple; but if it is then extended, the spasm instantly returns, making the limb rigid, and often completing the extension, just as the blade of a knife opens out under the influence of its spring, "clasp-knife rigidity." Occasionally, there occur brief flexor spasms, drawing the legs up. The knee-jerk is greatly exaggerated, and there can also be developed rectus-clonus and ankle-clonus. Electro-contractility is impaired early and gradually declines until abolished.

Seguin called attention to "a desire to micturate that is far less controllable than it should be in a healthy person."

The spastic gait is characteristic, termed by Hammond "the waddle;" the legs drag behind and are moved forward as a rigid whole, the toes catching against the ground, the patient showing a tendency to fall forward.

Sensation is unaffected. As the morbid process extends upward, the superior extremities suffer in the same manner as those of the lower.

Diagnosis.—The gradual development of weakness in the legs, excess of myotatic irritability, and spasms with developing spastic gait render the diagnosis clear. If the symptoms develop suddenly or acutely, the morbid condition is not of the degenerative variety.

Prognosis.—Complete recovery is rare. If the condition is early recognized, its progress may be held in check for a long time.

Treatment.—Rest is of great importance and every means should be taken to improve the general health. Massage and warm baths are of value. When the affection can be attributed to syphilis or mineral poisoning, increasing doses of potassium iodid or gold and sodium chlorid should be administered. Silver nitrate or silver oxid often retards the hyperplasia of connective-tissue. Benefit may sometimes follow the use of a weak galvanic current but as a rule electricity is disappointing in central diseases.

LOCOMOTOR ATAXIA.

Synonyms.—Posterior spinal sclerosis; tabes dorsalis.

Definition.—A chronic degeneration of the posterior columns of the spinal cord and the posterior nerve-roots, characterized by loss of coördination, neuralgic pains in the limbs, loss of sensation and reflexes, and visceral and trophic changes.

Causes.—The disease usually attacks males between the ages of twenty and fifty, one-half of the cases occurring between thirty and forty years of life. The most potent etiologic factor appears to be syphilis, although alcoholism, exposure, traumatism, etc., may be considered as contributory factors.

Pathology.—It may be considered as a general disease of the nervous system affecting both central and peripheral portions though mainly limited to sensory or afferent structures (Peterson). It is also described as a progressive destructive process which has a selective influence on certain tracts in the posterior columns with their roots and ganglia and to a less extent on the peripheral nerves, particularly the optic. The nerve-fibers of the cord are first involved. Their destruction is not a simple wasting, but is accompanied with evidence of irritation, such as swelling of axis-cylinders and, secondarily, proliferation of connective-tissue and slight congestion (Dana).

The degenerated portions of the cord appear grayish or slightly pinkish and translucent, and somewhat depressed. The pia is slightly thickened and may be turbid. Microscopically, the disease begins on either side in the posterior nerve-root and extends into the postero-external columns of Burdach while at higher levels the postero-internal columns of Goll are also involved. The posterior vesicular columns of Clark, the marginal zone of Lissauer, and the medullary bridge of Weigert may at times be affected. The morbid process usually begins and is most marked in the dorso-lumbar segment of the cord.

Symptoms.—Locomotor ataxia may be divided into three periods: 1, disturbances of sensation; 2, loss of coördinating power; 3, paralysis.

The onset of the disease is gradual, characterized by sharp, darting, electric-like pains in the lower limbs, with disorders of the gastrointestinal and genitourinary tracts. Associated with the pains is a loss of sensation in the feet, the patient being unable to distinguish between hard and soft substances in walking, and, if the upper portion of the spinal cord be affected, is unable to coördinate the muscles of the fingers sufficiently to button his clothing. A sensation of formication over the surface, especially over the lower limbs, and about the waist, the knee, and the ankle, is present; there is nearly always a feeling of constriction about the trunk — the girdle.

Loss of coördination, or ataxia, is manifested by the subject being unable to walk upon a straight line with his eyes closed, and with difficulty if his eyes are opened. There is inability to preserve the erect position with the feet close together, the body swaying widely and the patient falling on attempting to stand with closed eyes,— Romberg's symptom,— and as the malady progresses the patient throws his feet and legs in the most grotesque manner when walking. Although the patient is unable to coördinate the the muscles, their power is not lost, for, on being supported, he can kick or strike with his usual force.

The sight is early impaired, due to atrophy of the optic nerve, causing either double vision or inability to distinguish between different colors. Very early there is loss of pupil-reflex to light, the reaction to accommodation being preserved—Argyll-Robertson symptom. Ocular palsies may also occur. As the disease progresses, sensation becomes more and more blunted and pain is slowly recognized, frequently several minutes elapsing before the pricking of a pin is appreciated. A characteristic sign of the disease is the abolition of the patellar tendon-reflex—Westphal's symptom — as well as other reflexes in the lower limbs. Loss of the sensation of temperature also occurs. The electro-contractility is decreased in the affected limb. General emaciation is marked.

Vasomotor and trophic symptoms, more or less pronounced, occur in all cases. " Perforating ulcers " of the feet, circumscribed loss of hair, changes in the nails, and local sweatings are the more common. Muscular atrophy, either localized or general, is not infrequent.

Fränkel, under the term " hypotonia," describes a condition found in tabetic persons in which the patient, lying on a flat surface, can completely straighten his legs when at right angles to the body, which cannot be done by a normal man, whose knees will be bent when legs are at right angles with body.

Either early or late in the disease, occur disturbances in micturition and loss of sexual power and often desire. There also occur, in a fair number of cases, painless swelling and disintegration of various joints, particularly the knee and elbow — *the tabetic arthropathies*, or *Charcot-joints*.

At any period of the disease, peculiar *crises* or neuralgic attacks occur; if griping pains in stomach with vomiting, *gastric crises;* if renal pain or colic with disturbed urinary flow, *nephralgic crises;* if pain in bladder, *vesical crises;* if pain in rectum with hemorrhoids, *rectal crises;* if severe paroxysm of coughing, *bronchial crises;* if constriction of the throat with dyspnea, *laryngeal crises;* if cardiac pain and tachycardia, *cardiac crises.*

Paralysis finally ends the suffering of the patient. There is generally an entire absence of cerebral phenomena, although rarely delusions or dementia develop toward the end of the malady.

Diagnosis.—There are four pathognomonic symptoms of locomotor ataxia, the presence of which makes the diagnosis positive; they are Westphal's symptom — absence of patellar reflex; Romberg's symptom — swaying of body and inability to maintain erect position with closed eyes; the Argyll-Robertson symptom — loss of pupil reflex to light, but reaction to accommodation retained; Fränkel's symptom — hypotonia.

Chronic myelitis is characterized by paralysis, and the course of the affections are otherwise so different, that an error should be impossible.

Disease of the cerebellum presents symptoms of disordered coordination, but they are the result of vertigo, and are associated with headache, nausea, and vomiting, with absence of neuralgic pains and eye symptoms.

Paraplegia is a true paralysis, while locomotor ataxia is not. Neuralgic pain is not a symptom of paraplegia.

Gastralgia may simulate the gastric crises of locomotor ataxia, but the history and attendant phenomena will serve to make a distinction.

Multiple neuritis shows loss of power with pain and tenderness, but does not present the four pathognomonic symptoms mentioned above.

Prognosis.—The outlook is unfavorable. The disease runs a chronic and progressive course extending over several years with occasional remissions. Stationary periods may be encountered. In the early stages the progress may be retarded by treatment. The affection ultimately ends in death by some intercurrent disease.

Treatment.—Absolute rest, preferably in bed, over an extended period is essential to the proper management of each case. Excitement, mental exertion, and sexual excesses should be avoided. Measures should be taken to improve the general health independently of the nervous condition. For this purpose nutritious food, cod-liver oil, hypophosphites, strychnin, etc., are indicated. The association of syphilis with this affection calls for the administration of potassium iodid and the bichlorid of mercury in full doses. The chlorid of gold and sodium, gr. 1-20 (0.003 Gm.), three times daily, often serves to retard the progress of the disease. The best medicinal results are obtained from the use of silver nitrate, gr. ¼-½ (0.016-0.03 Gm.), or silver oxid, gr. ½ (0.03 Gm.), three times daily, withholding the drug at intervals of a few weeks to prevent discoloration of the skin (argyria).

Massage and systematic exercise are of great value. The system of Fränkel in which the patient is made to re-learn coördination and practice the same, from the most simple to the most complex movements, is undoubtedly productive of great benefit. Many modifications of this system are in vogue all of which are adapted to individual cases and differ little, if any, in principle. The employment of cold along the spine in the form of cold sponging, cold spinal pack, or short application of the cold douche is of value. The application of the galvanic continuous current along the spinal column with faradism to the wasting muscles is strongly advocated.

In the second stage, the suspension treatment of Charcot, has been followed by temporary improvement. It consists in the suspension of the patient during a period varying from one to four minutes, by means of the Sayre apparatus for applying the plaster jacket in spinal deformities.

The pains require rest in bed and the administration of analgesics. Counterirritation over the nerve-root supplying the painful part often relieves the distress. Massage and the alternate hot and cold douche are sometimes of benefit. The actual cautery applied to the back once a month is said to relieve the pains. The faradic brush, static spark, and anodal application of the galvanic current may be of value. Resort to opium or one of its derivatives, however, is nearly always necessary.

The various other symptoms should be treated on general therapeutic principles as they arise. When there is increased arterial tension nitroglycerin should be used, but its use must be guarded when aortic insufficiency is present. Cannabis indica is sometimes of value.

ATAXIC PARAPLEGIA.

Synonyms.—Combined lateral and posterior sclerosis; anterolateral sclerosis.

Definition.—A chronic degeneration of the lateral pyramidal tracts and of the posterior columns of the spinal cord, characterized by gradually developing paraplegia, with ataxia and spasms of the limbs.

Causes.—The causes are not so well determined as in other varieties of spinal sclerosis.

Pathology.—A sclerosis of the lateral and posterior columns of the spinal cord is a constant structural change. It is to be noted that the posterior columns show the morbid changes higher up than in locomotor ataxia,—the dorsal rather than the lumbar regions,—and that the root-zone of the postero-external column is much less involved. Nor do the lateral tracts show the same degree of involvement as in spastic paraplegia.

Symptoms.—The onset is slow and gradual, with loss of power in the lower extremities. The muscles involved are particularly the flexors of the thigh and knee. One leg may be weaker than the other. There is also ataxia, the patient being unsteady when standing with feet together (tabetic swaying), and he tends to fall if the eyes are at the same time closed. Spasms of the lower extremity gradually develop and finally become as marked as in spastic paraplegia. The knee-jerk reflex is increased, quick and extensive, and rectus- and ankle-clonus can be developed. The sexual power is lost early. Incontinence of urine is frequent.

Sensation is unimpaired, and neuralgic pains are absent, as are also eye symptoms.

Diagnosis.—The conditions, ataxic paraplegia is most liable to be mistaken for are locomotor ataxia and spastic paraplegia. The presence of knee-jerk and loss of power in lower extremities are of value in discriminating from locomotor ataxia. Spastic paraplegia is not associated with ataxia — indeed, ataxic paraplegia is spastic paraplegia *plus* incoördination.

Prognosis.—Unfavorable. The condition is progressive.

Treatment.—The same plan of treatment may be tried as recommended for lateral or posterior sclerosis.

CEREBROSPINAL. SCLEROSIS.

Synonyms.—Multiple sclerosis of the brain and cord; cerebral sclerosis; spinal sclerosis; disseminated sclerosis (Charcot).

Definition.—A degenerative disease of the brain and spinal cord; characterized by pains in the back, disorders of sensation, loss of coördination, tremor on motion, scanning speech, and some mental impairment.

Pathology.—The disease consists in the development of patches of grayish, translucent, tough nodules, varying in size from a microscopic object up to the size of a walnut, varying in number and widely distributed in the white matter of the hemispheres, ventricles, optic thalamus, corpus striatum, peduncles, pons, and cerebellum, while in the cord they are found in both the white and gray matter and in the columns. The deposits are also found in the nerve-roots and nerve-trunks. The nodules are composed of the neuroglia, much altered, and a newly-formed connective-tissue. The result of the growth of the nodules is pressure upon the nerve-structure, ending in its degeneration.

Symptoms.—The affection may be considered as of three varieties, depending upon the site of the most marked changes; cerebral, spinal, or mixed. The latter variety is the more common. It is observed in younger individuals than are the other forms of sclerosis.

The onset is usually insidious and is attended by more or less severe pains in the limbs and back, which the patient attributes to rheumatism, and a sensation of formication, itching, and burning in the limbs. Very rarely, the malady is ushered in with apoplectiform symptoms. Loss of coördination of the hands in writing, or of the feet in walking, soon becomes manifest, followed after a time by paresis, more or less general, with contracture of the muscles. Voluntary movements of the paretic limbs develop a tremor which subsides when the limbs are at rest,—*intentional tremor.* It is increased by excitement. It extends to the head and neck causing shaking of the head on raising it from a pillow or on similar movement. An early and frequent symptom is nystagmus. The loss of coördination, with tremor and with contractures of the muscles of the legs, gives rise to the "waddle" or "hop gait" when walking. The speech is slow, scanning, or slurring in character. There are also present headache, vertigo, and mental impairment together with an unnatural contentment of the feelings and with the surroundings. Disorders of vision from optic atrophy and disturbances of hearing may occur. Sexual impairment, vesical disorders, gastric and other crises, and bed-sores may also be symptoms of this condition. The knee-jerk and wrist-jerk are exaggerated and ankle-clonus is present. The disease is progressive, the symptoms developing as the various nerve-tracts are invaded. Trophic disturbances are seldom present.

The duration of the disease ranges from one to twenty years, the average being five to ten years. During this period the patient is very liable to develop pulmonary tuberculosis or chronic nephritis.

Diagnosis.—The following group of symptoms, characteristic of this disease, should prevent any error in diagnosis: pains in the limbs and back, loss of coördination in the feet and hands, muscular weakness with contractures, intentional tremor, nystagmus, scanning speech, disordered vision, increased reflexes, and vertigo.

Paralysis agitans may be mistaken for disseminated sclerosis. The chief points in the diagnosis are the presence in paralysis agitans of the fine tremor continually without shaking of the head, with a peculiar flexion and rigidity of the hand, while in cerebrospinal sclerosis the tremor is produced only on movement of the muscle, and is associated with shaking of the head. Paralysis agitans is a disease of middle life, sclerosis occurs under forty years. Changes in the voice, speech, and vision are present in cerebrospinal sclerosis, but absent in paralysis agitans.

Tumor of the pons or crus is accompanied by wild, jerky incoordination closely resembling disseminated sclerosis, but tumor also has headache, vomiting, optic neuritis, local spasm, and local paralysis.

General paralysis of the insane and disseminated sclerosis are frequently confounded, as are locomotor ataxia and primary lateral sclerosis. A careful consideration of the characteristic symptoms, already mentioned, will serve to make a distinction.

Prognosis.—Unfavorable. The disease slowly but steadily progresses, chronic nephritis or tuberculosis frequently developing and causing death.

Treatment.—There is no drug having the power to cure sclerosis. Syphilis has been the cause of the vast majority, if not all, of the cases observed by the writer, and potassium iodid, in large doses, or the following has seemed in a few instances to hold the disease in check for a time:

℞. Hydrargyri chloridi corros.	....gr. j	.065	Gm.
Liq. arsenici chloridi	...fʒ j	4.	Cc.
Inf. gentian.	.......ad f℥ iij	ad 90.	Cc.

M. S.—Teaspoonful three times daily, in water.

Attention to the general health and remedies to promote constructive metamorphosis will prolong life and add to the comfort of the individual. Massage, hydrotherapy, electricity, and systematic exercises may be of benefit and should be given a fair trial.

HEREDITARY ATAXIA.

Synonyms.—Friedreich's Disease; Hereditary Ataxic Paraplegia.

Definition.—A sclerosing disease of the lateral and posterior columns of the spinal cord which shows a predilection for certain families and occurs at an earlier age than locomotor ataxia.

Causes.—The etiology is obscure. The affection occurs in a number of the members of the same family and manifests itself between the ages of two and twenty-four.

Symptoms.—The essential features of this disease are ataxia and paraplegia. Pains are seldom present. Irregular jerky movements of the head, impaired speech, disordered vision, loss of muscular power, and diminished reflexes are common. Sensory phenomena are seldom marked and trophic disturbances are unusual. Deformities of the feet and lateral spinal curvatures are not infrequent.

Treatment.—The treatment is unsatisfactory. The disease tends to progress, although the course may be extremely slow and extend over several years. The measures recommended in locomotor ataxia are applicable to this affection.

SYRINGOMYELIA.

Synonym.—Syringomyelitis.

Definition.—A chronic disease of the spinal cord, characterized by the formation of cavities in the substance of the cord, associated with loss of the perception of pain and temperature over certain regions, and complicated with muscular weakness and atrophy, and, at times, trophic changes.

Causes.—The true cause is unknown. The affection is rare and occurs most often in males between the ages of ten and forty years. Hemorrhage and traumatism of the cord are believed to influence its production considerably.

Pathologic Anatomy.—There is present a tubular cavity or cavities in the substance of the spinal cord, the development of which is the subject of considerable discussion. It is thought that they may originate in either a faulty closure of one of the divisions of the primary central canal of the cord, for in the course of development the primary central canal of the cord becomes divided into two parts — an anterior and a posterior. The anterior division forms the permanent central canal. The walls of the posterior division gradually come together and form the posterior fissure. The imperfect closure of either of these divisions of the primary central canal may give rise to syringomyelia. Or, the abnormal cavity or cavities may depend on the disintegration of a gliomatous formation which originates generally in embryonal tissue about the central canal. The cavity varies in extent and location in different cases, and it is possible to find marked changes on autopsy which gave rise to no symptoms during the lifetime of the individual. The cervical cord is the usual seat of the disease.

Symptoms.—The condition develops slowly and insidiously, and is nearly always bilateral. There occur loss or diminution of the perception of temperature (heat and cold) and pain, the tactile sense being retained; and slowly developing muscular atrophy, due to involvement of the anterior horns of the cord. The atrophy usually affects the arm and shoulder of one or both sides, and it may begin in the hand. Associated with the muscular atrophy are muscular weakness and more or less fibrillary contractions. When the weakness involves the spinal muscles, scoliosis follows. Arthropathies occur in many cases, particularly involving the shoulder-joint. Trophic changes also involve the skin, often advancing to ulceration and even gangrene, and rarely to painless felons, such as occur in Morvan's disease. The general health of patients suffers but little in syringomyelia.

The disease is seen in many irregular types, the loss of temperature sense in one part and the loss of sensation of pain in another, and other irregular distribution of the characteristic phenomena. The symptoms of other forms of spinal disease, especially sclerosis, may be present in addition.

Diagnosis.—Progressive muscular atrophy is apt to be confounded with syringomyelia unless the changes in the temperature and pain senses are remembered. Morvan's disease is by many neurologists classed as a variety of syringomyelia.

Prognosis.—The affection is incurable, but the duration is rather long, extending over several years, often with periods of quiescence.

Treatment.—This is unsatisfactory and consists in measures for the relief of symptoms.

CAISSON DISEASE.

Synonym.—Diver's paralysis.

Definition.—An obscure condition occurring in divers and others working under increased atmospheric pressure, characterized by paraplegia, hemiplegia, anesthesia, or apoplectic attacks on return to the normal atmosphere. Increased atmospheric pressure is the sole cause. The manner in which the symptoms are brought about is not well understood. They usually subside in from several days to a few weeks. Occasionally death results.

Treatment.—The affection may be prevented to some extent by avoiding sudden changes in the atmospheric pressure and long-continued work under high pressure. The symptoms should receive the same treatment recommended under acute myelitis.

DISEASES OF THE NERVES.

SIMPLE NEURITIS.

Definition.—An inflammation of the nerve-trunks, characterized by pain, impaired sensation, motor paralysis, and atrophy.

Causes.—Among the principal causes may be included wounds, injuries, and compression of the nerves, extension of adjacent inflammation, exposure to cold and wet, rheumatism, gout, infectious fevers, syphilis, and lead-poisoning.

Pathologic Anatomy.—Hyperemia is the earliest change, and is soon followed by exudation into the nerve-sheath and connective tissue which becomes softened shortly and ultimately breaks down into a diffluent mass. The affected nerve is consequently red and swollen. The microscope shows that migration of white corpuscles takes place into the neurilemma and that the fibers have undergone more or less granular change. Recovery may take place before the nerve-elements are entirely destroyed, by absorption of the exudate. Inflammation of a nerve may extend upward (*neuritis ascendens*) or downward (*neuritis descendens*). In long-standing cases, the diseased nerves are found to be made up largely of connective-tissue, replacing the degenerated structure.

Symptoms.—The onset may be accompanied with febrile reaction. The most decided symptom is pain with tenderness along the course of the nerve-trunk and its peripheral distribution, of a burning, tingling, tearing, intense character, increased by pressure or motion. If the affected nerve be a mixed one,—sensory and motor,—spasmodic contractions and muscular cramps occur, fol-

[illegible]

[illegible]

Diagnosis.—Myalgia or muscular pain is not accompanied with paralysis, nor does the pain follow the course of a nerve.

Neuralgia has the pain, but as a rule, not the symptoms of neuritis.

Prognosis.—Generally favorable, with proper treatment.

Treatment.—The affected part should be placed at rest. Repeated blistering along the course of the nerve, preferably with the Paquelin cautery or a poker heated to a white heat, together with the administration of full doses of potassium iodid is usually successful in relieving the condition. Sedative liniments will also serve to lessen the pain. Sodium salicylate, phenacetin, and antifebrin may be of value at times, but in severe cases morphin, hypodermically, is necessary. In syphilitic cases, the iodids are indicated, and in those due to exposure to cold and wet and rheumatism, the salicylates and alkalies are of great value. In all cases, quinin sulphate, gr. ii–v (0.13–0.3 Gm.), every four hours, should be employed from the onset. As the acute symptoms subside, galvanism or a feeble, slowly-interrupted faradic current should be used to restore the functional activity of the affected nerve and the muscles to which it is distributed. Potassium iodid and strychnin during this period are also of value. If there are any manifestations of anemia, iron together with malt and the hypophosphites should be administered.

MULTIPLE NEURITIS.

Synonyms.—Polyneuritis; peripheral neuritis; disseminated neuritis; degenerative neuritis; pseudo-tabes; alcoholic paralysis; beri-beri (Brazil and India); kakké (Japan).

Definition.—*A parenchymatous inflammation of a number of symmetric peripheral nerves, simultaneously or in rapid succes-*

sion; characterized by pain, numbness, loss of power, or ataxia, with muscular atrophy. Mental symptoms are often associated.

Causes.—Multiple neuritis arises from a number of causes all of which are toxic in character and possess a predilection for the nerve-fibers. The principal poisons introduced from without that may induce the affection are alcohol, lead, arsenic, silver, mercury, phosphorus, anilin, benzine, carbon bisulphid, and ergot. The internal causes include the toxins of syphilis, leprosy, malaria, beriberi, acute infectious jaundice, diabetes, diphtheria, typhoid fever, septicemia, small-pox, rheumatism, gout, chorea, and cachectic states.

The affection occurs usually in adults between the ages of twenty and fifty. It may occur in children as a complication of acute anterior poliomyelitis and diphtheria. The female sex is most often attacked. Emotional disturbances, anemia, and exposure to cold may act as exciting causes. Alcoholic multiple neuritis is the most common form of the disease. Beri-beri and kakké are epidemic varieties of multiple neuritis.

Pathologic Anatomy.—The affection is generally bilateral and symmetric. An important characteristic is its peripheral distribution, the inflammation being most intense at the extremities of the nerves, lessening progressively toward the center, usually terminating before the nerve-roots are reached. The inflammatory process affects the nerve-fibers primarily and the sheath and connective-tissue secondarily — a parenchymatous inflammation. The affected muscles are paler and smaller than normal, the fibers being reduced in size and undergoing granular changes.

Symptoms.—The onset may be sudden, even overwhelming, causing rapid death, but is usually subacute or chronic from the beginning. According to the symptoms, the affection may be divided into three forms, motor, sensory, or ataxic.

The motor form manifests itself in motor weakness, chiefly involving the flexors of the ankles, the extensors of the toes, and the extensors of the wrists and fingers, situated in the forearm. Inflammation of the anterior tibial or peroneal nerve in the leg, and the radial branch of the musculo-spiral in the arm is common resulting in the double "foot-drop" and "wrist-drop" so characteristic of this disease. Any of the nerves of the body may be affected, the motor symptoms varying with the individual nerves.

Muscular atrophy begins early and progresses with the disease. The steppage gait is often observed.

The sensory form shows itself in pains, tenderness, tingling, and numbness with loss of cutaneous sensibility. At times the hyperesthesia of the extremities, especially the soles of the feet and the muscles, is so marked that the slightest touch cannot be borne.

The ataxic form is characterized by incoördination with or without sensory disturbances, but with loss of muscular sense.

These forms may exist combined to a greater or less extent. Atrophy of the muscles, feeble or absent knee-jerks, and absent or diminished electro-contractility are common to all forms. Trophic changes may occur in the nails, hair, and skin. A characteristic glossy condition of the skin with some edema often results from involvement of the vasomotor nerves. Rarely the vagus, optic nerve, and laryngeal nerve are attacked.

An *acute variety* of the disease may occur, in which the affection is ushered in with fever, 101°F.-103°F., rapid feeble pulse, headache, nausea, vomiting, and delirium or convulsions shortly followed by various combinations of the motor, sensory, and ataxic phenomena already described.

The *chronic variety* is unattended by febrile symptoms and begins insidiously with pains and other sensory disturbances, followed by weakness and wasting of the muscles, and the other characteristic manifestations of this disease.

Alcoholic multiple neuritis is attended by several characteristics which serve to distinguish it. Foot-drop is a typical symptom and there may be delirium, mania, and delusions associated with tremors. This variety usually affects all the limbs beginning in the flexors of the feet, being thus separated from the malarial form in which the legs are first involved; the diphtheria type, in which the pharyngeal and ocular muscles are first attacked; the rheumatic in which the muscles of the face are first affected; and the lead variety, which begins in the arms.

Beri-beri, or endemic neuritis, is an infectious form of multiple neuritis due undoubtedly to microörganisms. It is claimed that some defect in diet is a predisposing cause of this variety. The symptoms and development are much the same as seen in other varieties with the addition of edema and a tendency to effusions into the serous cavities and a cardiac involvement. In severe cases the pain is intense and the wasting rapid.

Diagnosis.—The distinctive features of this affection are its symmetric distribution, pain and tenderness over the nerve-trunks, peripheral nerves and muscles, the various sensory phenomena, and the loss of power with wasting of the muscles, beginning in the extremities. The history of some toxic condition will also aid in making the diagnosis. A careful consideration of these characteristics will serve to differentiate the condition from Landry's disease, neuralgia, locomotor ataxia, rheumatism, hysteria, poliomyelitis, and other affections with which it might be confused.

Prognosis.—The outlook is, as a rule, favorable, if early and proper treatment be instituted. Involvement of the respiratory muscles in acute cases may be the cause of a fatal termination. In long-standing cases, the probability of restoration of the affected muscles to normal is not very great.

Treatment.—The primary cause should be ascertained, and, if possible, promptly removed. Absolute rest in bed is of great importance. Pressure upon the affected parts should be carefully avoided. The parts should be wrapped in cotton-wool or flannel, and moist or dry heat, and sedative lotions or ointments should be applied. Temporary relief may be afforded by change in position of the limbs but unusual positions should not be long maintained on account of the possibility of contraction of the muscles and subsequent deformity. Antifebrin and similar preparations may be of benefit but in severe cases morphin, hypodermically, is necessary.

There is no specific medication for polyneuritis. In alcoholic cases, strychnin nitrate should be used; in malarial cases, quinin sulphate; in diphtheritic cases, tincture of the chlorid of iron and strychnin sulphate; in rheumatic cases, sodium salicylate, salol, or phenacetin; in syphilitic cases, mercury and potassium iodid; and in lead and other mineral poisonings, the iodids should be employed. In all cases, a generous nutritious diet with the administration of tonics is necessary.

During convalescence, moderate exercise, massage, and mild galvanism should be prescribed. Arsenic during this period is considered to be of great value as a constructive tonic.

NEURALGIA.

Definition.—A disease of the nervous system, manifesting itself by sudden pain of a sharp and darting character, mostly unilateral, following the course of the sensory nerves.

Varieties.—I. *Neuralgia of the fifth nerve;* II. *Cervicooccipital neuralgia;* III. *Cervicobrachial neuralgia;* IV. *Dorsointercostal neuralgia;* V. *Lumboabdominal neuralgia;* VI. *Sciatica;* VII. *Erythromelalgia* (Mitchell).

Causes.—The most important etiologic factors are adult life, female sex, heredity, anemia, malaria, syphilis, rheumatism, metallic poisons, gout, anxiety, mental exertion, exposure to cold and damp, injuries to the nerve-trunks, and reflex disturbances such as accompany eye-strain and dental affections.

Pathology.—The changes in the nerves are very vague. Neuritis is frequently present. The true nature of neuralgia is obscure. An impoverished condition of the blood, perhaps, underlies the affection.

NEURALGIA OF THE FIFTH NERVE.

Synonyms.—Tic-douloureux; Fothergill's disease.

Symptoms.—Paroxysmal pain, of a sharp, darting, stabbing character, most common at points along the course of the supra- and infra-orbital branches of the fifth nerve of the left side, attended with increased lacrimation, is characteristic of this affection. When of any duration, changes are observed in the nervous distribution, such as edema along the course of the nerve, gray eyebrows, and convulsive twitches of the muscles, termed "*tic-douloureux*," with tenderness at the infra- and supra-orbital foramina, as well as along the course of the nerve distribution.

CERVICOOCCIPITAL NEURALGIA.

Paroxysmal pain, of a sharp and lancinating, or deep, heavy, tensive character, along the course of the occipital nerve upon one or both sides, extending from the vertex, and on the neck as far down as the clavicle, and upward and forward to the cheek. It may be associated with hyperesthesia of the skin, and with cramps in the cervical muscles, and with attacks of herpes. A sensation of cracking at the nape of the neck is an annoying symptom in many cases.

CERVICOBRACHIAL NEURALGIA.

Paroxysmal pain of a severe, boring, burning, on tensive character, with sensations of numbness and weakness of the arm, hand, shoulder, scapula, and mamma, with tenderness along the cervical plexus. Edema of the arm and other parts along the distribution of the cervical plexus occurs if the neuralgia be of long duration, as the result of nutritive changes, the limb at times becoming pale, the skin glossy, dry, and harsh.

DORSOINTERCOSTAL NEURALGIA.

Paroxysmal pain of a sharp, and lancinating character, along the fifth and sixth intercostal spaces, often associated with the development of *herpes zoster*, or "*shingles.*" Tenderness is present at the points where the nerves emerge from the intervertebral foramina at the sides of the chest and at points in front.

LUMBOABDOMINAL NEURAGLIA.

Paroxysmal pain of a sharp and lancinating, at times heavy and dull, character, following the course of the iliohypogastric nerve, ilioinguinal and external spermatic nerve, supplying the integument of the hip, the inner side of the thigh, the scrotum and labium.

SCIATICA.

Paroxysmal pain following the course of the sciatic nerve usually as the result of a neuritis.

Symptoms.—Sciatica usually follows an attack of lumbago, the pain becoming fixed in the sciatic nerve; at times it is a true neuritis.

The pain is sharp, tearing, shooting, or lancinating in character, increased upon motion, shooting along the course of the nerve into the hip, inner side of the thigh, calf of the leg, ankle, and heel, at one or all of these points, in paroxysms lasting from a few hours to twenty-four hours or longer. Tactile sensation in the foot and motility in the limbs are impaired, and if of long duration, wasting of the limb occurs.

ERYTHROMELALGIA.

Synonyms.—"Red neuralgia."

Symptoms.—This form of neuralgia was first described by Dr. S. Weir Mitchell. The feet principally, are affected by intense redness and burning pain. For a considerable period before the condition is typically developed there are aching pains in the feet, particularly when used. The feet, in Dr. Mitchell's own words, "get redder and redder, the veins stand out in a few minutes as if a ligature had been tied about the limb, and the arteries throb violently for a time, until at length the extremities become of a dark purplish tint." As a rule, the redness only occurs when the feet hang down, and when at rest they may be pale and perspire freely. Blisters and ulcers follow slight contusions of the feet.

Diagnosis.—Erythromelalgia has been confounded with Raynaud's disease. The presence of pain, bright redness, throbbing, and increased temperature of the part are all the opposite of Raynaud's disease.

Prognosis of All Forms of Neuralgia.—The attack can usually be relieved, and in those cases in which the underlying cause can be ascertained and removed, the outlook is favorable for permanent cure. If the neuralgia is the result of the pressure of an exostosis, aneurysm, or other tumor, the prognosis is unfavorable. The variety known as erythromelalgia is very persistent. Fifth nerve neuralgia is likewise very obstinate to treatment.

Treatment of Neuralgia in General.—During the intervals between the attacks, the general health should be improved and all possible sources of reflex disturbance should be carefully removed. The diet should be highly nutritious and medication suitable for the individual case should be employed. In anemic patients iron and arsenic should be used; in rheumatic persons, the alkalies and salicylates should be given; in syphilitic cases or those due to mineral poisons, potassium iodid should be administered; and in the presence of malaria, quinin sulphate or hydrochlorate should be employed. Undue physical or mental excitement, exposure to cold and wet, and excesses of various kinds should be avoided. The following pill is of great value in all cases:

℞.	Quininæ sulphat.	gr. ij	.13 Gm.
	Morphinæ sulphat.	gr. 1-20	.003 Gm.
	Strychninæ sulphat.	gr. 1-30	.002 Gm.
	Acidi arseniosi	gr. 1-20	.003 Gm.
	Extracti aconiti	gr. ½	.032 Gm.

M.—Ft. pil. No. j.

S.—One every one, two, or three hours. (S. D. Gross).

All forms of neuralgia are more or less benefited with—

℞.	Quininæ sulph.	gr. iij	.2 Gm.
	Ferri reduct.	gr. j	.065 Gm.
	Acid. arseniosi	gr. 1-20	.003 Gm.
	Aconitiæ	gr. 1-120	.00054 Gm.

M. S.—In pill, every four or five hours.

The condition of the eyes, ears, nose, throat, and teeth should always receive careful attention. The presence of eye-strain, cerumen, adenoids, dental caries, etc., may be the origin of reflex disturbances that ultimately become neuralgias. The relation between them is not always apparent so that in all cases these structures should be routinely examined.

During an attack, the hypodermic injection of morphin sulphate and atropin sulphate, affords the most prompt and ready relief. Acetanilid, phenacetin, bromids, caffein, salicylic acid preparations and cannabis indica may also be used but are less efficacious. Moist or dry heat, chloroform liniment, menthol and chloral-camphor applications, acupuncture, and counterirritation may be employed locally.

In *trigeminal neuralgia,* the following combination is productive of great benefit:

℞.	Aconitinæ (Duquesnel)..gr. 1-10	.006 Gm.
	Glycerini,	
	Alcoholis ....āā fʒ j	āā 4. Cc.
	Aquæ menth. pip. ...ad f℥ ij	ad 60. Cc.

M. S.—Teaspoonful, repeated from four to eight times daily, carefully watched.

In *intercostal neuralgia,* the following is recommended:

℞.	Chloral ....ʒ j	4. Gm.
	Pulv. camphoræ ....ʒ j	4. Gm.
	Menthol ....ʒ j	4. Gm.

M.—Mix and rub together.

S.—Paint over painful parts with brush, as the occasion requires.

Facial neuralgia is often wonderfully benefited by the administration of fluid extract of gelsemium, ♏iii-v (0.2-0.3 Cc.), every three or four hours until its physiologic effects are produced. It may be combined with cannabis indica or belladonna. Excellent results often follow the use of *Moussette's pills* (aconite and quinin).

In sciatica, antipyrin, antifebrin, or phenacetin, gr. xx (1.3 Gm.), repeated two or three times daily may afford relief. Bartholow recommends deep injections of chloroform. Nitroglycerin may be of benefit, beginning with one drop of a one percent solution, three or four times daily, and gradually increasing the dose until four or five drops are taken several times daily. Mitchell advocates the application of a flannel bandage to the entire leg, changed daily, and a splint reaching from the axilla to the heel, held closely to the limb. This procedure insures absolute rest for the part. Rarely, this observer injects cocain hydrochlorate, gr. ¼ (0.016 Gm.), hypodermically, over the painful area. Tonics should also be employed. A spray of chlorid of methyl along the course of the nerve for a few moments often serves to relieve the distressing pain. Occasionally, the administration of full doses of potassium iodid and the application of a blister along the course of the nerve will be of benefit. Massage, acupuncture, nerve-stretching and electricity, and similar procedures may be tried in obstinate cases.

In *erythromelalgia,* medication has been of no avail. Rest and elevation of the limb affords relief in many cases. Mitchell recommends either nerve-stretching or in aggravated cases nerve-excision.

FACIAL PARALYSIS.

Synonym.—Bell's palsy.

Definition.—An acute paralysis of the seventh cranial,—the facial nerve, the great motor nerve of the muscles of the face,—the *nerve of expression.*

Causes.—Exposure to a current of cold air against the side of the face — over the *pes anserinus* — is the most frequent cause. It may also be due to injury or disease of the middle ear involving the nerve, tumor, blood-clot, or abscess in the cortical area or nucleus of the seventh nerve, or at the base of the brain, syphilis, rheumatism, or the infectious fevers.

Symptoms.—The facial nerve supplies the muscles of the face, the muscles of the external ear, also the stylo-hyoid, posterior belly of the digastric, the platsyma, one muscle of the middle ear, the stapedius, and one palate muscle, the levator palati; by means of the chorda tympani branch it controls the secretion of the parotid and submaxillary glands, and, possibly, the sense of taste. It also furnishes motor power to the azygos uvulæ, the tensor tympani, and the tensor palati muscles.

The onset is usually sudden, with tingling of the lips and tongue and upon looking into the mirror the patient is surprised by the perfectly blank, motionless side of his face; the corner of the mouth is depressed, the eyelids open, the face drawn toward the well side, and the patient is unable to expectorate, whistle, or swallow.

Any of the muscles innervated by the nerve may participate in the paresis.

The electro-contractility is feeble or lost. The reflexes are abolished. If there is loss of taste in the anterior portion of the tongue, it indicates involvement of the nerve in its passage through the temporal bone.

Diagnosis.—Facial paralysis, such as accompanies hemiplegia and similar affections is attended by normal reflex excitability and cerebral symptoms due to involvement of other nerves. Facial palsy, in the presence of otorrhea, imperfect hearing, obliquity of the uvula, and loss of taste, is due to a lesion of the nerve in the aquæ ductus Fallopii. The peripheral form of Bell's palsy is complete and the taste is normal and the uvula straight.

Prognosis.—In cases of peripheral origin the outlook is favorable. In others, it depends entirely upon the character of the underlying cause and the ease with which it may be removed.

Treatment.—In peripheral facial neuritis, the bowels should be opened thoroughly and the salicylates administered. Diaphoresis should be obtained by the hot bath and pilocarpin or diuresis by means of potassium acetate and diluents. Blisters should be applied in front of the ear. As the acute symptoms subside, potassium iodid and strychnin should be given and galvanism and massage should be applied to the affected muscles. In cases, due to middle-ear disease special treatment is necessary. In paralysis of central origin, the iodids may be employed, but apart from this, medication is of little avail.

GENERAL NERVOUS DISEASES.

CHOREA.

Synonyms.—St. Vitus' dance; insanity of the muscles.

Definition.—A functional disorder of the nervous system; characterized by irregular spasmodic fibrillary movements of groups of muscles, with weakness, more or less approaching paralysis of of the affected parts. Excitement increases these movements, while sleep causes their cessation.

Causes.—It is essentially a disease of childhood, and its production may be greatly influenced by female sex, rheumatic diathesis, habit, neurotic temperament, heredity, mental excitement, spring season, and reflex disturbances such as produced by adherent prepuce, masturbation, worms, dentition, eye-strain, etc. The affection may be observed at times during pregnancy and after hemiplegia.

Pathology.—There are no constant lesions. Emboli are believed to be the cause in some cases. The affection is believed by many observers to be a neurosis and by others an infection.

Symptoms.—The onset is usually gradual, the child seemingly grimacing or jerking the arm or hand, as if in imitation, followed soon by decided irregular jactitations of the muscles of the face (histrionic spasm), of the eyelids (blepharospasm), eyeballs (nystagmus), and the shoulder, arm, and hand, finally extending to the lower extremities, interfering greatly with motility; in severe cases there is inability on the part of the patient to feed himself or to hold anything in the hands. The speech is often unintelligible, the tongue constantly moving in an irregular manner.

The heart's action is tumultuous and irregular, associated often with a soft, blowing, systolic murmur, most distinct at the base. The muscles are usually quiet during sleep, although this is not always the case. The mind is somewhat blunted, the temper irritable, and the memory impaired. If the irregular muscular movements are confined to one side of the body, it is termed *hemichorea.*

Rheumatism and endocarditis may occur as complications or as sequels.

Diagnosis.—Chorea was confounded with epilepsy until the points of distinction were pointed out by Sydenham.

Huntington's or *chronic chorea* is distinctly hereditary, and, instead of being fibrillary contraction of muscles, involves whole groups of muscles, so that the patient seems to be posturing and grimacing, with a dancing movement, with many queer contortions of the face and head. Generally, all the muscles of the body are involved. It may have associated the fibrillary muscular contractions of St. Vitus' dance.

Paralysis agitans has general muscular tremor, beginning in one limb, gradually progressing, uninfluenced by treatment; it is a disease of the elderly.

Post-hemiplegic chorea is the choreic movement of a paralyzed limb.

Chorea insaniens is characterized by violent movements preventing ordinary voluntary movement and attended by fever, delirium, and exhaustion sometimes ending in death. It occurs most often in adults.

Prognosis.—The vast majority of cases recover, but relapses are very frequent.

Treatment.—The child should be removed from all excitement, mental and physical, and placed at comparative rest among the best hygienic surroundings. Many cases improve rapidly when confined to bed in a darkened room. The diet should be light and the secretions should be rendered free. The cause should be removed if possible. All reflex irritation such as accompanies eye-strain, intestinal parasites, dental disorders, adherent prepuce, etc., should receive appropriate attention.

Arsenic is the most reliable remedy yet introduced for the treatment of this affection. It should be pushed until its first physiologic effects present themselves, after which the dose should be gradually reduced until all the symptoms disappear. The best preparation for use in this connection, is the solution of the arsenite of potassium (Fowler's solution), ♏v (0.3 Cc.), increased to ♏x (0.6 Cc.), or even ♏xv (1 Cc.), three times daily. Fluid extract of cimicifuga, ♏xx-fʒi (1.3-4 Cc.), three times daily, is of value, especially in cases following rheumatism. Those cases resisting arsenical medication may rapidly improve under hyoscyamin hydrobromate, gr. 1-200 - 1-100 (0.00032-0.00065 Gm.), three times daily. Obstinate cases occasionally respond to antipyrin, gr. x (0.6 Gm.), four times daily. Quinin is also of benefit at times. In anemic individuals iron should be administered. If the muscular movements interfere with sleep, recourse should be had to hyoscin, bromids, chloral, or morphin sulphate.

EPILEPSY.

Definition.—A chronic disease, of which the characteristic symptom is a sudden loss of consciousness, attended with more or less general convulsions.

Causes.—True epilepsy almost always arises first, during the growth and development of the brain. Heredity exercises a very strong predisposing influence. The family history in many cases contains records of insanity, epilepsy, hysteria, and similar conditions in the relatives of the patient. Worry, anxiety, depression, fright, syphilis, uterine disease, brain-tumor, and meningeal thickening may at times be etiologic factors. Reflex irritation from intestinal parasites, eye-strain, etc., may induce epileptoid convulsions which if long-continued may bring about true chronic epilepsy. The affection usually manifests itself before puberty and seldom begins after twenty-five years of age.

Pathologic Anatomy.—There are no constant anatomic lesions, as yet, associated with essential epilepsy.

In "Jacksonian," "cortical," or "partial epilepsy," however, the "motor cortex" is irritated by disease and there occur tonic and clonic spasms of the same character as in general epilepsy, confined to a single arm, or an arm and half the face together, or may be the entire half of the body. These epileptiform attacks furnish precise data as to the locality of the lesion; spasms affecting the distribution of the facial nerve point to the lower third of the central convolution; of the arm, the middle third of the central convolution; of the lower extremity, the upper third of the central convolution.

Varieties.—I. *Epilepsia gravior*, le grand mal; II. *Epilepsia mitior*, le petit mal.

Symptoms.—*Le grand mal* is preceded by a more or less pronounced and curious sensation, the so-called *aura epileptica*.

The attack proper is sudden, the subject suddenly falling, with a peculiar cry, loss of consciousness, and pallor of the face, the body assuming a position of tetanic rigidity, succeeded after a few moments by more or less pronounced clonic convulsions, followed by coma of several hours' duration. The subject awakens with a confused or sheepish expression, with no knowledge of what has occurred, unless he has injured himself during the attack, either by the fall, or, what is very common, has bitten his tongue during the convulsions. When the convulsions follow each other in rapid succession without any intervening periods of consciousness, the condition is termed *status epilepticus*. The convulsive outbreaks may be followed by maniacal attacks or the condition known as *epileptic automatism*, during which various acts are performed unconsciously by the patient.

Le petit mal is manifested either by attacks of vertigo, the consciousness being preserved, or by a passing absent-mindedness, either form being associated with slight convulsive phenomena followed by slight coma, or mental confusion of short duration.

The mental functions are not, as a rule, injured by attacks of epilepsy, unless they recur very frequently. Indeed, when at wide intervals, the subject seems relieved by them, "the sudden, excessive, and rapid discharge of gray matter of some part of the brain on the muscles," the so-called "electric storm," having cleared the cerebral atmosphere.

The great majority of epileptics suffer from chronic gastric catarrh, and have at the same time an inordinate appetite (boulimia); indeed, an attack of gluttony may immediately precede a fit. The liability of patients suffering from epilepsy to develop tuberculosis and nephritis is very great.

Diagnosis.—*Uremic convulsions* closely resemble epileptic attacks, but the dropsy or general edema and albuminous urine, and increased temperature of the former should guard against error.

Hysteria or feigned convulsions may mislead the most practical expert. In convulsions of this character, however, consciousness is seldom completely lost; the patient is never injured in any way; the temperature, pulse, and respiration remain normal; arching of the back occurs (opisthotonos is absent in epilepsy); and the duration is longer. In epilepsy, there is primary pallor of the face which is followed by a dusky, livid, and swollen appearance during the convulsion, this being replaced by ordinary congestion during the period of coma. Relaxation of the sphincters is common in attacks of *grand mal.*

Organic brain disease may be distinguished from epilepsy by the occurrence of its convulsions at a much later period in life, the character of the convulsions, and the history of injury, syphilis, etc. Jacksonian epilepsy begins as a spasm of a limb or some portion of a limb, and is confined there, or may gradually extend until even a general convulsion occurs.

Prognosis.—In idiopathic epilepsy the prognosis is unfavorable; the vast majority of cases will not be arrested by treatment, but the frequency and severity of the attacks will be greatly diminished. Epileptoid convulsions of reflex origin such as sometimes accompany intestinal parasites, eye-strain, etc., usually recover promptly when the cause is removed.

Treatment.—*The Attack*: To avert an impending attack inhalations of amyl nitrite, ♏iii-v (0.2-0.3 Cc.), or a few whiffs of chloroform, or the hypodermic injection of morphin sulphate may be employed. Hyoscin and chloral may also be used. These remedies are also indicated after the onset of the convulsion. Means should be taken to prevent the patient seriously injuring the tongue; to this end a portion of a towel, or a long piece of wood such as a clothes-pin, or the handle of a tooth brush should be inserted between the teeth. Small objects so used are dangerous. The patient should also be prevented from otherwise injuring himself and on the subsidence of the convulsion should be placed comfortably at rest in a quiet room.

Status epilepticus is always a dangerous condition, and efforts to prevent it should be made by active medication the moment a series or group of fits occur. The following combinations sometimes are wonderfully successful in aborting the status:

℞.	Chloral	gr. xxx	2. Gm.
	Tinct. cannab. indicæ	♏xv	1. Cc.
	Infus. digitalis	f℥ j	30. Cc.

M. S.—By high enema, repeated if indicated in two or three hours.

Dr. Spratling, Craig Epileptic Colony, recommends:

℞.	Tinct. opii deodorat.	♏v	.3 Cc.
	Potassii bromidi	gr. xxx	2. Gm.
	Chloral	gr. xx	1.3 Gm.
	Liq. morph. (U. S.)	♏iss	.03 Cc.
	Aquæ	f℥ ss	15. Cc.

M. S.—By mouth, or, if unable to swallow, by enema.

A hypodermic injection of morphin sulphate, gr. ⅓ (0.02 Gm.), and atropin sulphate, gr. 1-60 (0.001 Gm.), has sometimes broken up a series of epileptic spasms.

The Interval: During the interval, the patient should be carefully examined to determine the character of any exciting causes. Reflex disturbances of all kinds should be promptly removed by treatment. The diet must be carefully regulated, excluding or allowing to be used very moderately, meats, tea, and coffee. Alcohol and tobacco should be interdicted. The skin, kidneys, and bowels should be kept in normal condition by appropriate measures. Moderate exercise is of great value. When the patient's general condition is below normal, iron, arsenic, quinin, and cod-liver oil are indicated. Strychnin is contraindicated as it increases the tendency to convulsive attacks.

In addition to the measures already mentioned, the frequency and severity of the attacks may be greatly lessened by the internal administration of potassium bromid in doses sufficient to abolish the faucial reflex and to produce symptoms of bromism. Combinations of the several bromids are equally efficacious and less irritating than the potassium salt. Any tendency towards the formation of acne pustules during the administration of the bromids may be combatted by the addition of one drop of Fowler's solution to each dose. When for any reason the bromids are inapplicable, silver nitrate, belladonna, or cannabis indica may be used. In syphilitic cases, potassium iodid should be given in addition. In all cases the treatment must be continued for at least two years after the last attack.

Gowers highly recommends the following in cases complicated with cardiac dilatation:

℞. Potassii bromidi	........gr. xx	1.3 Gm.
Tinct. digitalis	.........♏x	.6 Cc.

M. S.—Three times a day, well-diluted.

The following is the combination used in the insane wards of the Philadelphia Hospital:

℞. Sodii bromidi,		
Potassii bromidi	....āā ʒ iv	āā 16. Gm.
Liq. potassii arsenitis	...fʒ iss	6. Cc.
Aquæ menthæ pip.	.....f℥ iij	90. Cc.
Inf. gentian. comp. q.s.	ad f℥ viij	ad 240. Cc.

M. S.—Tablespoonful, diluted, three times daily.

Brown-Séquard's mixture for epilepsy is as follows:

℞. Potassii iodidi	8 parts.
Potassii bromidi	8 "
Ammonii bromidi	4 "
Potassii bicarb.	5 "
Inf. columbo	360 "

M. S.—One teaspoonful before meals, and three dessert-spoonfuls on going to bed.

The following is an effective combination for the "mixed bromids":

℞.	Sodii bromidi	℥ j	30. Gm.
	Potassii bromidi	ʒ vss	22. Gm.
	Ammonii bromidi	℈ viij	10.6 Gm.
	Potassii bicarb.	ʒ ij	8. Gm.
	Inf. columbo	f℥ x	300. Cc.
	Aq. chloroformi .q. s. ad	Oj	ad 480 Cc.

M. S.—Tablespoonful equals gr. xxx (2 Gm.).

Surgical intervention may be of value in some cases of Jacksonian epilepsy, but in general it is somewhat disappointing in its results.

HYSTERIA.

Definition.—A functional disorder of the nervous system, of the nature of which it is impossible to speak definitely; characterized by disorders of the will, reason, imagination, and the emotions, as well as motor and sensory disturbances.

Causes.—A morbid condition confined principally to women. Young girls, elderly single women, widows, and childless married women are the most frequent subjects of the disorder. The paroxysms frequently develop during the menstrual epoch. The menopause is another frequent period for its manifestations. A peculiar condition of the nervous system, either inherited or acquired, is responsible for the phenomena of hysteria, the peculiar manifestations being excited by disturbances of either the sexual, digestive, circulatory, or nervous systems.

Hypochondriasis, a peculiar mental condition, characterized by inordinate attention on the part of the patient to some real or supposed bodily ailment or sensation. A continual introspection, as seen in males, is a condition much like the hysteria of the female.

Pathology.—The true nature of the affection is unknown. Structural changes are never found except when the condition complicates some organic disease.

Symptoms.—These will be considered under the headings of the *hysteric paroxysm,* and the *hysteric state.*

The Hysteric Paroxysm or fit occurs nearly always in the presence of other individuals and develops gradually with sighing, meaningless laughter, causeless moaning, nonsensical talking, and gesticulations, or a condition of fidgets followed with a sensation of choking, dyspnea, and a ball in the throat—the *globus hystericus*. These and similar symptoms precede the fit, during which the unconsciousness is only apparent, the patient being aware of what is transpiring about her. During the paroxysm the patients may struggle violently, throwing themselves about, their thumbs turned in and their hands clenched. Again, spasmodic movements occur, varying from slight twitching in the limbs to powerful general convulsive movements, and to almost tetanic spasms.

The paroxysm ends by sighing, laughing, crying, and yawning, and a sensation of exhaustion. During the attack it will be noted that the surface and face are normal, showing absence of respiratory embarrassment, the breathing varying from very quiet to spluttering and gurgling sounds, the pupils not dilated, the pulse normal, the temperature normal, and absence of foaming at the mouth and wounding of the tongue.

The Hysteric State is shown by disturbances of the mental and sensory-motor functions respectively. It may be a permanent condition or occur at intervals with greater or less severity.

Mental disturbances. The patients are emotional, erratic, excitable, impatient, and self-important, showing marked defects of will and mental power.

Sensory disturbances. These consist of either a condition of exaggerated sensibility or hyperesthesia, as shown by the marked effects from the slightest irritation and the cutaneous tenderness along the spine, or a condition of anesthesia, as shown by the apparent absence or recognition of pain after severe irritation, or a perverted sensibility, as shown by the feeling of tingling, numbness, and formication. The anesthetic area is ischemic. Sensibility to heat or cold is often absent. There is great perversion of the special senses in many of the cases. Severe pain at the top of the head, as if a nail were being driven in it, is peculiar to hysteria and is termed *clavus*.

Charcot, referring to the ovarian hyperesthesia of hysteria, says: "It is indicated by pain in the lower part of the abdomen, usually felt on one side, especially the left, but sometimes on both, and occupying the extreme limits of the hypogastric region. It may be extremely acute, the patient not tolerating the slightest touch; but in other cases pressure is necessary to bring it out. The ovary may be felt to be tumefied and enlarged. When the condition is unilateral, it may be accompanied with hemianesthesia, paresis, or contracture on the same side as the ovarialgia; if it is bilateral, these phenomena also become bilateral. Pressure upon the ovary brings out certain sensations which constitute the *aura hysterica,* but firm and systematic compression has frequently a decisive effect upon the hysterical convulsive attack, the intensity of which it can diminish, and even the cessation of which it may sometimes determine, though it has no effect upon the permanent symptoms of hysteria."

Motor disturbances. These phenomena embrace every variety of motor disturbance, from exaggerated excitable movements to defective or complete loss of power. With the paralysis that may occur, neither nutrition nor sensation is constantly impaired. Hysteric paralysis is liable to frequent and sudden changes, the loss of power often disappearing suddenly. Aphonia, from paralysis of the laryngeal muscles, is a frequent form of paresis. Some hysteric patients refuse to even make an attempt at speech (mutism). Hysteric contractures often are most extensive and persistent. Under some emotion or unknown cause a group or groups of muscles contract abruptly or by degrees, the spasms involving flexors or extensors or both with changes in reflexes, and lasting for days or years. Convulsive seizures are common.

A curious enlargement of the abdomen is observed sometimes, constituting the so-called *phantom tumor.* This region presents a symmetric prominence in front, often of large size, with a constriction below the margin of the thorax and above the pubes. The enlargement is quite smooth and uniform, soft, very mobile as a whole from side to side, resonant, but variable on percussion, and not painful. Vaginal examination gives negative results, and under chloroform the prominence immediately subsides, returning again as the patient regains consciousness.

Among the numerous other symptoms that may develop in a hysteric patient are disturbances of digestion, circulation, and respiration, and disorders of micturition and menstruation.

Among other phenomena that belong to the Hysteric State are to be mentioned *hystero-epilepsy,* a condition of hysteria to which is superadded the convulsion, epileptic in form; *Catalepsy,* a condition in which the will seems to be cut off from certain muscles, and in whatever position the affected member is placed, it will so remain for an indefinite time. There may or may not be unconsciousness and loss of sensation; *Trance,* the individual lying as if dead, circulation and respiration having almost ceased; *Ecstasy,* a condition in which the individual pretends to see visions, and acts in a most ridiculous manner.

Diagnosis.—The hysteric state is so general in its manifestations that it is to be borne in mind in diagnosing all ailments occurring in women. The diagnosis is attended with great difficulty, however, and requires the display of all the skill of the clinician to prevent error.

Prognosis.—Death from either a hysteric fit or the hysteric state is the rarest of events, if it ever occurs. The ultimate recovery of a hysteric patient is of frequent occurrence. Marriage has cured many cases, although it can hardly be advised by the physician.

Treatment.—For the hysteric attack, little need be done, as a rule, unless the paroxysm is violent or prolonged, in which case valerianate of ammonia, Hoffman's anodyne, or aromatic spirits of ammonia may be administered. In severe cases, Charcot recommends making firm pressure over the ovarian region.

The management of a confirmed case of hysteria will tax the skill of the most astute physician. It is in connection with hysteria that the peculiar phenomena supposed to arise from applying different metals to the surface of the body have been noticed.

Moral and hygienic measures are of the first importance in the management of hysteric patients. The treatment of hysteric patients by isolation is strongly urged by many specialists. Dr. S. Weir Mitchell has devised a plan for bedfast hysteric patients, of massage, faradization, and forced feeding, which has been successful in a number of cases.

There is no fixed therapeutic treatment for hysteria, the various symptoms calling for interference as they arise. It is well, however, to avoid the use of stimulants, and opiates, chloral, and other sedatives.

NEURASTHENIA.

ynonyms.—Spinal irritation; nervous prostration; nervous ex- stion.

efinition.—A debility of the nervous system, causing an in- lity or lessened desire to perform or attend to the various duties occupations of the individual. It is a purely functional con- ion.

Bartholow describes it as consisting "essentially in an exaggerated susceptibility to bodily impressions and false reasoning thereon."

Causes.—Heredity, neurotic temperament, sexual excesses, alcohol, tobacco, mental exertion, emotion, overwork, and various chronic diseases are the principal causes. Men are especially liable to the affection.

Symptoms.—Nervous debility may affect any organ of the body. It is a condition of nerve-tire or exhaustion, and hence the nervous energy necessary for functional activity of any particular organ may be wanting, a fair example being seen in cases of nervous dyspepsia.

One of the earliest manifestations of nervous exhaustion is an irritability or weakness of the mental faculties, as shown by inability to concentrate the thoughts, and efforts to do so causing headache, vertigo, restlessness, fear, and a feeling of weariness and depression, together with an army of symptoms attendant on general nervousness.

There may be ocular disturbances, cardiac palpitation, coldness of the hands and feet, and chilliness followed by flashes of heat, followed in turn by slight sweating. Patients are troubled with insomnia, or fatiguing sleep, accompanied with unpleasant dreams.

In the male there are genitourinary disorders, with pains in the back, giving the dread of impotence. In females, painful menstruation, ovarian irritation, and irritable uterus.

The "neurasthenic stigmata" are: Feeling of pressure on head; disturbance of sleep; pain in back; muscular weakness; dyspepsia; sexual disorders, and mental disturbances.

Diagnosis.—It is of importance to determine between a true nervous exhaustion and nervous debility the result of organic disease.

A study of the history of the case, together with the symptoms, should prevent error.

Neurasthenic symptoms in puberty are strongly indicative of mental instability, and great care must be exercised to prevent actual insanity from developing.

Prognosis.—Usually some mental weakness remains after recovery from an attack of neurasthenia.

Treatment.—The physician should remember that neurasthenia is not a disease *per se,* but that the victim is a sick individual needing the best environment, rest, and good food. Attention to the secretions, diet, and surroundings, with rest and diversion of the mind are essential to success. Travel, short of fatigue, pleasant companionship, and relief from responsibility should be advised. Bathing, massage, and galvanism are important aids in the management. In anemic and weak individuals the rest-cure proposed by Dr. S. Weir Mitchell will be of value.

Among the internal remedies that are of value in this condition may be mentioned arsenic, strychnin, valerianate of zinc, phosphorus, fluid extract of cocoa, cocoa wine, and the compound syrup of hypophosphites. Quinin sulphate, in small doses, gr. i-ii (0.065-0.13 Gm.), daily, for weeks, seems to lessen the excitability of the nervous system. The following is an excellent tonic in this affection:

℞.	Acid. phosphoric. dil. . . . fʒ ij	8. Cc.
	Ext. cocæ fld. . . . . fʒ vj	24. Cc.
	Tinct. nucis vomicæ . . . . fʒ ij	8. Cc.
	Syr. zingiberis . . . . f℥ iss	45. Cc.
	Aquæ menthæ pip. . . ad f℥ vj	ad 180. Cc.

M. S.—Tablespoonful after meals, in water.

RAYNAUD'S DISEASE.

A very rare disease, characterized essentially by symmetric gangrene. The cause is unknown. The affection is associated with some disturbance of the vasomotor system, as a result of which local stagnation of the peripheral circulation occurs. In consequence of this there arise localized anemia, congestion, and finally gangrene, symmetrically distributed. The condition is observed most often in neurotic women under thirty years of age and in children. Pain is a prominent symptom. The treatment is unsatisfactory; the attacks continue but the prognosis as to life is favorable. Local warmth, friction, and galvanism together with the internal administration of tonics are indicated.

EXOPHTHALMIC GOITER.

Synonyms.—Graves' disease; Basedow's disease. Parry's Dis.

Definition.—A disease of the nervous system, characterized by protrusion of the eyeballs, enlargement of the thyroid gland, dilatation of the arteries, and palpitation of the heart, tremor, + excessive perspiratio

Causes.—An undemonstrable condition of the nervous system, either inherited or acquired, is the predisposing cause of Graves' disease. Among the exciting causes are anemia, shock, fright, chagrin, worry, and reverses of fortune. It is more frequent in women than in men. Probably excessive secretion of Thyroid.

Pathologic Anatomy.—"Some structural alterations have been found, in a majority of cases, in the sympathetic ganglia, and especially in the inferior ganglia." (Bartholow.) The veins and arteries of the thyroid gland are dilated, the result of a vasomotor paralysis. The enlargement of the gland is the result of the dilated vessels, and a serous infiltration of its tissues, followed, if long-continued, by hypertrophy. A considerable increase of fat behind the eyeballs has been observed. In the majority of cases more or less anemia exists.

Symptoms.—The development of the group of symptoms may occur suddenly, as the result of some great shock to the nervous system, but in the majority of instances they develop slowly and insidiously, with cardiac palpitation, with paroxysms of more marked acceleration, or tachycardia, the pulse-rate varying from 90 to 120, 150, and rarely as high as 200 beats per minute, and soon pulsations of the vessels of the neck and thyroid gland may be felt and seen. The enlargement of the thyroid gland—the goiter—appears gradually after the development of the circulatory disturbances, although rarely it may be the first symptom observed. The goiter is elastic, rather soft, and has a thrill similar to an aneurysm. The degree of enlargement varies in different cases, and in none ever attains a very great size. Following the development of the goiter occurs the protrusion of the eyeball,—the

x Gut sympt. exactly opposite in Myxoedema = where there is diminished Thyroid secretion exophthalmos,—which may be confined to one eye, but usually occurs in both. Prominence of the eyeball may be the first symptom observed, but usually it does not develop until after the appearance of the goiter. The degree of protrusion varies from a slight staring expression to a point so great that the eyelids cannot cover the balls. Associated with the protrusion of the eyeballs is incoördination in the movements of the eyelids and the eyeball, the sign of Graefe, so that when the eyes are quickly cast down, the eyelids do not follow them, the sclerotic being visible below the upper lid. Diminution in the power of convergence during accommodation (Möbius' symptom) and widening of the palpebral angle (Stellwag's sign) may also be present. Vision is unimpaired. Conjunctivitis may arise, the result of the imperfect protection of the protruding ball by the eyelids.

Associated with the pathognomonic symptoms are nervousness, irritability of temper, headache, insomnia, vertigo, fits of despondency, aphonia and cough the result of pressure of the goiter, disorders of digestion, increase of temperature, anemia, and loss of flesh.

Diagnosis.—The fully-developed disease presents no difficulties in diagnosis, but during its incipiency, before the characteristic symptoms have appeared, the disease may be confounded with such conditions as cardiac disease, neurasthenia, lithemia, malaria, or incipient phthisis.

Prognosis.—Recovery occurs in a fair number, but, as a rule, the course is slow and protracted. The disorders of the circulation lead to dilated heart in many cases, and ultimately death occurs from this cause. Relapses are frequent.

Treatment.—One of the first injunctions to be placed on a case of exophthalmic goiter is rest, both physical and mental, as well as freedom from worry or emotional excitement; little progress will be made if this point be neglected. The associated anemia requires the administration of tonics such as iron, arsenic, etc., and nutritious, easily-digested food. To control the circulatory disturbances digitalis and strophanthus are of inestimable value. In my own experience, most satisfactory results have followed the use of tincture of strophanthus, ♏v (0.3 Cc.), from three to six times daily. Silver nitrate, gr. ⅛ (0.008 Gm.), after meals is of value, alternating with strophanthus or digitalis. Bartholow em-

x Thyroidectin Tabs (P D & Co) are of dried ~~[illegible]~~ blood of an animal from whom the thyroid has been remov
Gives good results in Exophl Goitre. Average dose gr v ti

ploys quinin, ergotin, and belladonna, in combination, and obtains beneficial effects. Extract of thyroid gland effected rapid and complete recovery in three cases under my own observation using it in three grain (0.2 Gm.) doses three times daily. The initial dose of this drug should always be small. Galvanism to the cervical sympathetic and pneumogastric is always a beneficial adjunct to the medicinal treatment. Surgical intervention may be considered in the failure of other methods after a fair trial. The general nervousness, restlessness, and insomnia will often call for special treatment, when use may be made of chloral, potassium bromid, sulphonal, or trional. It is better, however, not to use this class of drugs in a routine manner, but for the special indications only. x

TETANY.

Synonyms.—Tetanilla; intermittent tetanus.

Definition.—A succession of tonic, usually bilateral, painful muscular spasms, particularly of the extremities, occurring at irregular intervals, without loss of consciousness.

Causes.—The true cause is unknown. Some cases are believed to be infectious; others are produced reflexly as in the case of those attacks associated with lavage, removal of the thyroid gland, pregnancy, etc. It is usually seen in rachitic children and young neurotic adults. Heredity, emotion, hysteria, and the infectious fevers are also etiologic factors. The disease is rare in America. The pathology is obscure.

Symptoms.—Tetany consists in the occurrence of intermittent spasms in the muscles of the arms, hands, legs, or feet, or, rarely, the face and larynx (laryngismus stridulus), associated with pain or "cramp." The hands are thrown into a position such as they assume in writing, or such as is taken by the hand of a midwife; or the hand may be tightly closed, or one or more fingers may be cramped. The elbows and shoulders may be affected at times. In the feet the toes are drawn down and the instep upward, as in equinus. The knees may be cramped or the legs extended. Any of the muscles may be involved. Trousseau pointed out that in those suffering from tetany, pressure upon the affected extremities at certain points will excite the spasms. The duration of the spasms varies from a few moments to several hours, the intervals being from an hour to a day or more. A certain periodicity is noticed as to the hour of the day or night. The electro-contractility is increased, as are also the reflexes. Erb first described the peculiar galvanic exaltation found in this disease. The consciousness is always preserved, although the patients are very nervous.

Diagnosis.—Tetanus and tetany may be confounded, and yet trismus is rare in the latter, and always present in the former.

Prognosis.—Favorable. Cases induced by lavage and removal of the thyroid may prove fatal.

Treatment.—There is no special treatment. The secretions and excretions should receive attention and a normal body-tone should be maintained. Potassium bromid, gr. xx-xl (1.3-2.6 Gm.), well-diluted, three times daily, is often of value. Urethan, gr. x (0.6 Gm.), every three or four hours, is also highly recommended. Gowers advises digitalis for the painful cramps in the calves that occur in the early morning hours (*nocturnal tetany*). Gray has observed excellent effects follow the application of cold to the extremities and ice to the spine. In all cases when the symptoms are very severe it may be necessary to resort to sedatives such as morphin and hyoscin.

OCCUPATION NEUROSES.

Synonyms.—Professional neuroses; artisans' cramp.

Varieties.—Writers' cramp; piano-players' cramp; telegraphists' cramp; violin-players' cramp; dancers' cramp.

Definition.—A group of affections of the nervous system, characacterized by the occurrence of spasm (cramp) and pain in groups of muscles, in consequence of over-use or frequently-repeated muscular acts.

Causes.—Undetermined. It has been noticed that many persons suffering from occupation neuroses have a neurotic family history.

Symptoms.—The manifestations of any of the several varieties of this condition generally develop slowly with a sensation of stiffness in the used member, the part feeling fatigued and heavy, eventually being incapacitated for work by the occurrence of spasmodic contractions. Attempts to move the part produce pain and often tremor. Actual paralysis may be present. There is often the sensation of prickling and numbness in the affected member. The electro-contractility is preserved until atrophy from non-use develops. Associated with the local changes there are nervousness, mental worry, and often depression.

Diagnosis.—The history of the case and its results make the diagnosis easy.

Prognosis.—The outlook is often unfavorable. Treatment should be long-continued as the prognosis is uncertain. Obstinate cases often recover with persistent treatment. Recurrences are not uncommon.

Treatment.—The affected part should be placed at absolute rest. General rest with mental quietude is also beneficial. The general neurotic condition of the patient should receive attention. Locally, massage, friction, faradism, and passive movements are very efficacious. The following combination has been employed with success:

℞.	Zinci phosphidi	.........gr. ij	.13 Gm.
	Ext. nucis vomicæ	......gr. x	.6 Gm.
	Ferri albuminat.	.......gr. xxx	2. Gm.

M.—Ft. pil. No. xxx.
S.—One after meals.

PARALYSIS AGITANS.

Synonyms.—Shaking palsy; Parkinson's disease.

Definition.—A nervous disease of unknown pathology, characterized by tremors, progressive loss of power in the affected muscles, moderate rigidity, with alterations in the gait, and at times mental impairment.

Cause.—Age seems to be an etiologic factor, most cases developing after fifty years. It is most frequent in women.

Pathologic Anatomy.—No characteristic lesion has as yet been determined. It being a disease of past middle life, there is probably an interstitial hyperplasia of some layer of the cortex, from alterations in the intima of the vessels.

Symptoms.—The onset is gradual, the tremor beginning in one of the extremities, most often the hand and forearm. At first it can be controlled by the will, for a time at least, and is suspended by voluntary movement. The disease gradually extends until an entire side or the upper or lower limbs are involved. The face and head rarely present tremors, but are not exempt. Facial expression is lost and speech is slow and somewhat measured. A peculiar rigidity of the affected muscles is characteristic of the advanced stage. "At this stage of the disease the hands are apt to assume the so-called bread-crumbling position, *i. e.*, the thumb and the fingers approximate and move restlessly over one another, as in the act of crumbling bread. There is often a tendency on the patient's part to go forward,—so-called propulsion,—and this is sometimes so marked that if the patient is once started in a walk forward, his gait becomes more and more rapid, and he cannot stop himself" (Gray). The patients are usually restless and annoyed with insomnia. The general health is fair. The mind is generally retained, although melancholia and mild dementia have been noted in a few cases.

Diagnosis.—*Disseminated sclerosis* has a tremor, but only on voluntary movements—intention tremor. There is also scanning speech and ataxic gait, with mental enfeeblement, as shown by an unnatural contentment with the physical condition and surroundings.

Chorea possesses a tremor, but the movements are general, and partcularly involve the muscles of the face. Again, chorea is a disease of children and young adults.

Prognosis.—Complete recovery is very rare. Improvement often results from early treatment. The disease does not tend to shorten life, but its course is indefinite.

Treatment.—Physical and mental rest are necessary in all cases. Nutritious diet, cod-liver oil, hypophosphites, arsenic, and iron are necessary to restore and maintain the general health. Friction, massage, bathing, galvanism, and specially-arranged gymnastics are of great value in this condition. Drugs such as hyoscyamin sulphate, gr. 1-30 - 1-10 (0.002-0.006 Gm.), three times daily, and hyoscin hydrobromate, gr. 1-200 - 1-100 (0.00032-0.00065 Gm.), three times daily, are often of benefit.

MYXEDEMA.

Definition.—A progressive disease characterized by an infiltration of the connective-tissue with a gelatinous substance, general failure of the health, and mental failure, due to or associated with atrophy of the thyroid gland.

Cretinism is considered akin to myxedema, save that it is a congenital condition associated with alteration or absence of the thyroid gland.

Cachexia strumipriva, a condition following the extirpation of the thyroid gland, especially in the young, gives symptoms resembling myxedema.

Causes.—The cause of the atrophy of the thyroid gland is unknown. It is more frequent in women than in men and usually develops about middle life. The disease is said to have followed the extirpation of the gland in the adult.

Morbid Anatomy.—Atrophy of the thyroid gland, sometimes more advanced in one lobe than the other is constant. "The pituitary body has been found increased in size" (Wood). Until the functions of the thyroid gland are more fully understood, the steps in the changes resulting from its atrophy can not be explained.

Symptoms.—The disease develops slowly, often a number of years elapsing before all the characteristic phenomena are present. The face and neck, and often other parts of the body, have a bloated appearance. The normal wrinkles are obliterated, the nose is wide and thick, the lips thick and everted, the mouth enlarged, as is also the tongue, giving a coarse and broadened or mask-like appearance to the features. The skin is denser and does not pit on pressure, but is pale or chalk-like, or yellowish-white, with often a small reddish patch on either cheek. The expression of the countenance is immobile and stupid. The hands and feet are enlarged, the skin is coarse and dry. The shape of the hands is changed, presenting a "spade-like" appearance. The mental condition is sluggish and stupid, with loss of memory and of interest in the environments and affairs of life. Occasionally hallucinations of sight occur. The tendency is toward a dementia. Patients often complain of neuralgic pains and numbness and a sense of muscular weakness. The temperature is always below the normal. Anemia develops, and often a subacute nephritis or a glycosuria or phthisis follows.

Diagnosis.—*Dropsy* or a general edema has a superficial likeness to myxedema, but a study of the symptoms should prevent error, as pitting on pressure does not occur in myxedema.

Prognosis.—Under treatment a great improvement can be produced but whether a permanent cure results is not yet fully determined.

Treatment.—The body-surface should be protected from cold by warm clothing. Warm bathing followed by inunctions of olive oil is also beneficial. Warm climates are best adapted for these patients. The administration of thyroid extract, beginning with gr. ss (0.03 Gm.) after meals, gradually increasing the dose until several grains are taken daily or until symptoms of thyroidism appear, is of great value. The remedy should be continued over a long period and withheld when evidences of thyroidism such as nervousness, restlessness, insomnia, dyspnea, rapid pulse, cardiac palpitation, gastrointestinal disorders, confusion of mind, or delirium become manifest. The anemia and muscular weakness are overcome by the use of iron, strychnin arseniate, gr. 1-60 (0.001 Gm.), and nuclein.

ACROMEGALY.

Acromegaly is a nutritional disease of unknown origin characterized by marked enlargement of the osseous and soft structures particularly of the face, hands, and feet. It occurs usually in males, developing in most cases before the age of thirty and is associated with disease of the pituitary body. In addition to the structural enlargements, there are headache, polyuria, spinal curvature, disorders of the special senses, headache, and various neurotic symptoms. The condition is incurable. The course is chronic and may extend over several years, death ensuing from some intercurrent disease.

Treatment is of little avail. Extracts of pituitary body, thyroid gland, spleen, or bone-marrow may be administered.

MENTAL DISEASES.

MELANCHOLIA.

Synonyms.—Depression of spirits. psychalgia.

Definition.—A variety of mental alienation, characterized by more or less profound depression, with either no marked intellectual disturbance or the presence of more or less incoherence, and hallucinations and delusions. The cerebral mechanism develops a condition of supersensitiveness, all impressions being exaggerated, and a state of abnormal self-consciousness existing.

Varieties.—Melancholia simplex; hallucinatory melancholia, melancholia agitata; melancholia attonita; hypochondriac melancholia; chronic melancholia; senile melancholia.

Causes.—Heredity, failing health, grief, domestic and financial worries, neurasthenia, menstrual irregularities, pregnancy, childbirth, lactation, climateric, gastrointestinal disorders, alcoholic and sexual excesses, and organic brain disease may be mentioned as causes. Religion rarely causes this form of insanity although it frequently lends color to it. It is most frequent in women and in the young. Attacks of melancholia are more frequent in the spring and early summer months, and statistics show that suicides also are more frequent during these periods.

Pathology.—The alterations in the nerve-structure, underlying an attack of melancholia, are undetermined. Anemia and sluggish nervous energy are constant phenomena, but are hardly the only conditions disturbing the cortex.

Symptoms.—Melancholia may be the initial stage of a mania, delusional insanity, or paretic dementia, or a stage of *folie circulaire*.

Mental: The cardinal condition is a feeling of depression, misery, or mental anguish or pain, for which no adequate cause may exist. The onset is usually gradual, with a disposition to neglect duties and self, the patient worrying over a something he cannot explain. The world is dark and gloomy, with a foreboding of some awful calamity that is to affect or wreck the patient or his family. Suspicion, distrust, and often, fear of wife, children, relatives, or friends are common. Insomnia is a constant and stubborn symptom. The memory is maintained, and the reasoning faculties are usually intact. The patient may sit quietly, declining or unable to talk (silent melancholia, or mutism), or be restless, according to the character of the emotions affected.

Physical: The patient presents either an anxious or a woebegone expression. Headache, particularly a post-cervical ache, is a very constant symptom. The skin is dry and harsh, the respirations superficial, the cardiac action slow and feeble and there are gastric catarrh, constipation, and scanty, high-colored urine. The tongue is flabby and coated, and the appetite is poor. The refusal to take food is most characteristic.

Hallucinatory melancholia is an aggravated form of the disease, in which in addition to the painful mental reflexes, there are distressing hallucinations and illusions, the patient living in a realm of terror. The attack may be the result of a delusion, but much more frequently the depression and foreboding give rise to the delusion. The delusions of melancholia are usually of self-accusation, self-abasement, and justified persecution; the patient feels that he is being punished for some transgression, imaginary or otherwise.

The manias of persecution and the monomanias of suspicion are all of a melancholic type, the result of painful hallucinations.

Hypochondriac melancholia shows all subjective impressions with disturbed memory, leading to the belief that the bowels have been removed, food cannot be digested, that the brain has turned around, that the blood cannot circulate, and that gallons of blood have been drawn from the body. These distressed individuals are often conscious of every organ of the body and experience disagreeable impressions coming from them all, and as a consequence are irritable, fretful, and exacting. It is to be remembered that not uncommonly these patients really have an organic disease affording a foundation for the delusions.

Melancholia agitata is that variety characterized by continual agitation, in which the fearful and distressful thoughts and imaginations cause wringing of the hands, restless walking, rhythmic swaying of the body, and prayers beseeching help, with tears flowing down their cheeks, crying out for assistance and protection. Incoherence and violent impulses are frequent, the excitement often resembling an attack of mania.

Melancholia attonita, or melancholia with stupor, is marked by the patients seeming to be overwhelmed, sitting mute, motionless, and expressionless, refusing to assist themselves in any way, and often requiring mechanical feeding. Memory is usually impaired in this variety, and attacks of violence may occur.

Chronic melancholia is the continuation of the depression over a long period, the individual living in the fear of impending danger or punishment for supposed acts, for long periods of time, often with apparent lucid periods.

Senile melancholia is a condition of extreme mental distress associated with beginning senile dementia.

Suicidal impulses are present in a fair proportion of cases of melancholia, and unless there is everlasting vigilance the patient will succeed in his insane desire.

Diagnosis.—The cases of simple melancholia are readily determined. *Melancholia agitata* is frequently mistaken for acute mania. *Melancholia attonita* closely resembles acute dementia—a condition, it is but fair to mention, denied by many alienists.

Prognosis.—A typical attack of melancholia runs a definite course, not unlike the typical course of a fever. It is favorable in the mild cases of all forms not associated with organic disease, and in those who have not reached the climacteric. Delusional melancholia has the most unfavorable prognosis. Pronounced cases of melancholia attonita are more apt to terminate in dementia than any other variety.

Treatment.—Change of environment and rest are essential. Attention to the gastrointestinal canal is of the greatest importance, as the dyspepsia and constipation of melancholic patients is a barrier to their recovery. Frequent bathing, with friction to the surface, aids in the eliminative action of the skin. The diet must be of the most nutritious character. If food is persistently refused, mechanical feeding must be practiced. The late Dr. Gray was a strong advocate of small doses of opium, or morphin, in acute melancholia, and in properly selected cases it is a most valuable agent. Tincture of quebracho, ʒ j-ij (4-8 Gm.), well-diluted, three times daily, is often a valuable remedy. If the arterial tension is relaxed, good results follow the use of digitalis. Sodium phosphate is often useful.

Many cases of melancholia seem to be due to a brain fatigue, and if the patient can be given many hours' sleep in the early days of the attack recovery is assured. In melancholia attonita, excellent results often follow the use of cannabis indica in increasing doses. Such tonics as quinin, arsenic, and strychnin are of value in building up the patient and as the strength improves open-air exercise must be employed. Insomnia must be combatted by evening bathing and feeding and by the use of chloral sulphonal, trional, or hyoscin.

MANIA.

Synonyms.—Insanity; madness.

Definition.—An intense mental exaltation, with great excitement, loss of self-control, with, at times, absolute incoherence of speech, and loss of consciousness and memory. (Clouston.)

Mania is a condition characterized by an abnormal exaltation and activity of the mental functions,—the intellectual faculties, the emotions, and the will,—and may show itself by irrational talking and acting, by delusions, illusions, and hallucinations, and by unusual muscular activity or movements. (Chapin.)

A mental condition in which there is an emotional exaltation, accompanied by illusions, hallucinations, delusions, great mental and physical excitement, and a complete loss of the inhibitory power of the will; in acute cases, and frequently in chronic forms of the disease, there is marked destructiveness and a tendency to violence. (Wood.)

An attack of mania may be *acute, subacute,* or *chronic.*

Causes.—Inflammation or other organic disease of the brain or its membranes, mental shock or strain, domestic, moral, or financial worry, excesses of various kinds, ovarian disease, menstrual disorders, climacteric in neurotic individuals, pregnancy, parturition, lactation, anemia, alcoholism, syphilis, and hereditary predisposition are the most frequent causes.

Pathology.—There are no constant morbid changes associated with mania. In all varieties of acute insanity there exists vitiated nervous energy or impaired vitality, the result of over-excitement or over-stimulation, motor disturbance, or autoinfection, due to the imperfect elimination of the products of tissue-waste.

If death follows the acute symptoms, the vessels of the brain and membranes are engorged, but in the majority of instances the brain-structure is normal.

If death occurs in chronic mania, the most frequent change found will be thickened and adherent dura mater. Any form of organic change may be found *post-mortem* in those dying of any form of mania.

"There is no reason why a mere dynamic brain disturbance should not kill and leave no structural trace, any more than that it should for months abolish judgment, affection, and memory, and then pass off and leave the brain and all its functions intact." (Clouston.)

Symptoms.—Acute Mania: The onset may be abrupt, or follow a period of emotional depression, associated with lassitude, feeling of unrest, disinclination to work, and disorders of the gastrointestinal canal, with insomnia and an introspection; these symptoms constitute the *melancholic stage* of mania.

The *maniacal stage* is characterized by loud talking, intense egotism, violent motions of the limbs and body, great restlessness, and excitement; the thoughts flow with wonderful freedom and amazing rapidity, the condition often resembling the symptoms of early alcoholic intoxication; as the exaltation continues the patient becomes either sullen, irritable, and angry, offering violence to those around him, or he becomes garrulous, talking of his personal affairs, is confidential and communicative to strangers, often making egotistic offers, passing frequently into incoherence of language and action. Sexual passions are frequently exalted, and acts of masturbation practiced, with outbreaks of vulgar, obscene, and profane language, which is entirely foreign to the individual in mental health. Delusions are an almost constant symptom, of a superficial or transitory character, changing with every new appearing mood. The maniacal patient is sleepless, or may have short naps, at once continuing his chatter on awakening.

Any attack may show all of the symptoms mentioned, or any one or more of them, but the great majority of cases show *intense egotism, loud talking, violent motion of limbs or body, hurry, excitement, insomnia, incoherence, and incessant noise.*

The course of an attack shows periods of remissions and exacerbations, with nocturnal crises; loss of flesh and mental weakness are often marked as the attack progresses.

Acute delirious mania, typhomania, is a psychosis of sudden onset, attended with increased bodily temperature, dry tongue, quick, feeble pulse, scanty urine, and marked by delirium with sensuous hallucinations, marked incoherence, restlessness, refusal of food, loss of memory, and rapid bodily wasting, terminating frequently in death.

Amenorrheal mania consists of attacks of mania occurring at the menstrual epoch. Homicidal, suicidal, and various hysteric impulses are frequent.

Mania-a-potu is an attack of acute delirium, due to alcoholic excesses in those engaged in a sudden debauch, or who have drunk heavily and eaten little, for a comparatively short period.

Asthenic mania is that form in which there is general anemia associated with neurasthenic symptoms.

Dancing mania is an hysteric mental state in which, through sympathy and imitation, dancing of a most grotesque and extravagant character occurs. It is usually epidemic.

Delusional mania is the result of fixed delusions, either causing or associated with, the maniacal outbreak.

Erotic mania, erotomania, presents systematized delusions of an erotic character, not necessarily accompanied by animal sexual desire. *Nymphomania* is a morbid, irresistible impulse to satisfy the sexual appetite, and is peculiar to the female sex.

Epileptic mania follows an epileptic paroxysm, and is often of a most violent kind, the maniacal acts being of the most treacherous and malicious character.

Hallucinatory mania presents visual, auditory, olfactory, and other sense hallucinations.

Homicidal mania is any variety of mental disease in which there is a desire or an attempt on the part of the patient to commit murder. The condition may be the result of delusions that the persons attacked either are persecuting or going to kill the patient, or of the excessive excitement that vents itself in destructiveness, combativeness, or desire to kill; or there may be a morbid desire, impulse, or craving to do murder; or the homicidal act may be unconsciously done during an acute delirium, or a paretic or epileptic maniacal impulse. In cases of murder the question of responsibility, or the difference between the insane criminal and the criminal is not always readily determined. With insane criminals, in the *act itself* lies the satisfaction and not the object, while with criminals the *act* is only a means to an end; to the former, crime is a pleasure, to the latter a paying business, necessitating, it may be, disagreeable or horrible acts.

Morphinomania is the insane craving for the stimulating action of morphin—a moral insanity.

Puerperal mania is the maniacal outbreak as seen in the puerperal woman. This is now thought to be of septic origin, although the mental strain through which the female has been passing is a predisposing factor in those who have a neurotic history.

Transitory mania, or ephemeral mania, is a rare form of maniacal excitement of sudden onset, violent and decided in character, accompanied by great insomnia, incoherence, and more or less complete unconsciousness of familiar surroundings. The attack as suddenly terminates, the duration being from a few hours to a few days.

Senile mania is the mental exaltation occurring in persons with senile arterial changes or senile cerebral atrophy. It is soon followed by dementia.

Recurrent mania, or chronic mania with lucid intervals of longer or shorter duration. This is generally of alcoholic origin.

A maniacal outbreak may present any one or a number of the varieties named.

Chronic Mania: A condition of continual mental exaltation, the acute symptoms having continued in a chronic course. The line that distinguishes between an acute and chronic mania must always be somewhat arbitrary and unscientific. The duration of the mania beyond twelve months is usually considered sufficient to determine the condition, and this is well, since it precludes the possibility of terming the condition incurable. If the term chronic mania was restricted to those cases in which, between the exacerbations of restlessness, excitement, and destructiveness, were evidences of dementia, less confusion would occur.

Terminations of Mania.—About fifty percent of acute manias, not due to organic disease, recover after periods varying from one month to several years. A fair proportion of cases make a partial recovery and are able to return to their work, but always showing some alteration in character or affection, or some eccentricity, or a slight mental weakness. About twenty percent of cases terminate in dementia or mental death and this is always the fear in each case. Two percent of cases die, either the result of exhaustion or from the organic condition causing or associated with the attack.

Prognosis.—The question of recovery, partial or complete, is always difficult to determine, depending upon the cause, temperament, disposition, education, nationality, and the normal mentality of the individual. Recovery is usually gradual; rarely sudden restoration occurs.

Favorable indications are: sudden onset, short duration, youth of patient, absence of fixed delusions, good appetite, increasing hours of sleep; moderate or no increase in temperature, pulse, and respiration; no evidences of mental weakness, no paralysis or alteration of pupils or articulation, no epilepsy, no unconsciousness to the calls of nature, and no former attacks. Unfavorable indications are the opposite of these, the presence of organic brain disease, a strong hereditary tendency, and the possession of an excitable disposition or nervous diathesis.

Treatment.—The indications for treatment are to quiet the exalted mentality and to promote the constructive metamorphosis. Every means should be used to lessen the excitement of the patient and produce refreshing sleep. A hot or warm bath is frequently one of the most soothing means of reducing excitement; changing the environment of the patient and placing him under the care of a good, firm, but kind and intelligent nurse is of importance; the society of the family or friends must be forbidden, for visits act as stimulants to the disordered intellect and encourage discussion on the part of the patient as to the character of the treatment, and thus reduce the discipline so essential to early recovery.

If means of this character are unavailing, and, unfortunately, in the majority of attacks they will be, then resort must be had to sedatives, for every day's continuance of the maniacal outbreaks lessens the chances of restoration. Hyoscin hydrobromate, gr. 1-120 - 1-60 (0.00032-0.001 Gm.), repeated two or three times daily watching its effect on the pupils, sulphonal, gr. xx (1.3 Gm.) repeated with caution, chloralamid, gr. xxx-xl (2-2.6 Gm.) repeated three or four times daily, and trional, gr. xxx (2 Gm.) repeated in two or four hours are of great value in this connection. The latter is one of the most reliable drugs for relieving maniacal excitement and insomnia. Tincture of passion flower (passiflora incarnata), ʒi-ʒii (4-8 Cc.), several times daily may also be used. When there is much excitement and the pulse is weak, full doses of the bromids and digitalis are of benefit. If the muscular excitement is pronounced, good results follow the use of morphin sulphate, hypodermically, alone or combined with either atropin sulphate, hyoscin hydrobromate, or duboisin sulphate.

In attacks of acute mania with flushed face, throbbing arteries, full pulse, and delirious excitement, fluid extract of gelsemium, ♏ii (0.12 Cc.), every hour until dilatation of the pupils and ptosis develop or until improvement occurs, is indicated. Tincture of veratrum viride, ♏ii-v (0.12-0.3 Cc.), is also useful under such circumstances.

Ice or cold to the head is likewise beneficial in cases with flushed face and throbbing temporals. Post-epileptic excitement is best controlled by large doses of chloral given by mouth or rectum.

The general condition of the patient calls for the most prompt and efficient treatment. Attention to the gastrointestinal canal and kidneys is of paramount importance, as many attacks of mania are the result of autointoxication from the retention of the products of mal-assimilation and tissue-waste. The diet should be of the most nutritious character, peptonized or hot milk, hot broths, eggs, and often alcoholic or malt liquors, administered at frequent intervals.

Patients not infrequently refuse food on account of lack of appetite, abhorrence of food, or from fear of poisoning, when recourse must be had to the naso-stomachic tube, or nutritive enemata. If the breath be heavy, the tongue badly coated, the bowels costive, and the skin sallow, the very best results follow washing out the stomach, providing the maniacal condition permits. Tonics are of great value, a combination like the following always being beneficial:

℞.	Quininæ sulphat. ....gr. xlviij		3.1	Gm.
	Strychninæ sulphat. .... gr. ss		.032	Gm.
	Acid. hydrochlor. dil. ..fʒ iij		12.	Cc.
	Aquæ chloroformi .....f℥ iij		90.	Cc.
	Aquæ menthæ pip. q.s. ad f℥ vj	ad	180.	Cc.

M. S.—Dessertspoonful, diluted, every four or six hours.

The question of removal to a hospital for the insane arises in nearly all cases, and should in my judgment be answered, in the vast majority of instances, in the affirmative, as the discipline, regular hours, and order of a well-managed hospital for the insane have a most remarkable effect on the majority of patients.

EPILEPTIC INSANITY.

Definition.—A mental condition caused by or the result of epilepsy.

Causes.—The careful study of the brain of those having epileptic insanity has failed to determine why some epileptics suffer from any of the insanities and others have their normal mentality, and another group are better after a convulsion.

I am familiar with ten cases of epilepsy all of whom seem much brighter, mentally, after their paroxysm, but in whom, after a drinking bout, each epileptic attack is followed by a wicked homicidal mania of many weeks' duration.

Varieties.—Pre-epileptic mania; post-epileptic mania; dementia epileptica; imbecility with epilepsy.

Symptoms.—The mental changes constituting epileptic insanity, save in the cases of epilepsy with imbecility or idiocy, develop after some years of the ordinary epileptic paroxysms.

Pre-epileptic mania consists in attacks of mania some days or hours preceding the epileptic convulsion. The patient is morose, irritable, and threatening, often making homicidal attacks on those around him, friends or foes. Rarely the epileptic seizure is replaced by various insane or so-called hysteric acts, as fits of dancing, laughing, crying, screaming, swearing, or scolding.

Post-epileptic mania follows the epileptic paroxysm, either taking the place of the comatose state or following it. The maniacal acts during these outbreaks are often of the most desperate and impulsive character, many an asylum physician and attendant carrying scars the result of attacks of post-epileptic maniacs.

Epileptic dementia is the terminal mental obliquity resulting in about thirty percent of insane epileptics who do not succumb previously to nephritis or tuberculosis.

Epileptic imbecility is a congenital condition in which epilepsy and imbecility are associated.

Prognosis.—The great majority of persons suffering from epileptic insanity develop, sooner or later, either nephritis or tuberculosis. Recovery from epileptic mania is a rare occurrence, although I am familiar with two cases. Thirty percent ot epileptic maniacs progress to dementia in from five to ten years.

Treatment.—There is no doubt but that full doses of the bromids lessen the severity and frequency of the paroxysms. If the attack can be anticipated, it may sometimes be averted by an enema of chloral, gr. xx-xxx (1.3-2 Gm.), or chloralamid, gr. xl-lx (2.6-4 Gm.), or amyl nitrite, ♏v (0.3 Cc.), by inhalation or by mouth. For the condition of status epilepticus the following combination, alternated with saline purgatives, has given good results:

℞.	Chloral	gr. xx	1.3 Gm.
	Tinct. cannab. indicæ	♏xv	1. Cc.
	Inf. digitalis	f℥ j	30. Cc.

M. S.—Administer by enema every three or four hours.

The use of opium for a long period has broken up the recurrent maniacal attacks in several patients under my care.

The general condition of the patient must receive careful attention, as there is a strong tendency to the development of nephritis, tuberculosis, and gastric catarrh. This class of patients are great feeders—often gluttons—and are sure to eat more than they can properly assimilate. Free action of the bowels and kidneys must be promoted.

Never contradict, nor attempt to reason with, an epileptic during the period of excitement.

CIRCULAR INSANITY.

Synonym.—Folie circulaire.

Definition.—A mental disease characterized by regularly alterciated with anemia, arsenic and strychnin seem to be valuable. Two cases were rapidly improved with small doses of hyoscin hydrobromate, gr. 1-300 - 1-200 (0.00022-0.00032 Gm.), morning and evening. When food is refused by the insane, and stomachic or nasal tube or rectal feeding is necessary, the stage of food refusal is often wonderfully shortened by adding sulphonal, gr. x-xv (0.6-1 Gm), to each feeding.

DELUSIONAL INSANITY.

Synonyms.—Delusional mania; delusional melancholia; primary delusional insanity.

Definition.—A mental state, with fixed or partly systematized delusions, associated with either brain exaltation or excitement *without* maniacal acts, or a mental depression, minus the somatic symptoms of melancholia.

An insane delusion is a false belief for which there is, or may be, no reasonable foundation and which would be incredible under the given circumstances to the same person if of sound mind, and concerning which his mind is not open to permanent correction through evidence or argument.

An insane delusion is a belief in something that would be incredible to sane people of the same class, education, or race, as the person who expresses it; this resulting from diseased working of the brain-convolutions.

Causes.—Cerebral and bodily exhaustion, the result of overwork, neglect of personal hygiene, or alcoholic, tobacco, drug or sexual excesses, impairment of the nerve-centers consequent to fevers or shock, the climacteric period, worry, and insufficient food are the most common causes.

Pathology.—The affection runs a subacute or chronic course and seldom ends directly in death, usually being terminated by some intercurrent organic disease. In the few cases in which post-mortem examinations have been made, the vessels of the brain were found torpid or dilated, due in all probability to a vasomotor paresis which gave rise during life to an imperfect cerebral circulation.

Symptoms.—Either following an attack of acute mania or melancholia, but more commonly without either of these conditions, occurs a set delusion or delusions, which, to the patient, are so real that no amount of argument can dispel his or her belief in them. These cases are often classed as manias or melancholias, but, as they do not run the ordinary course of either of these conditions, they are best classed clinically by themselves. The acuteness or subacuteness of the attack distinguishes them from paranoia. Among the almost endless variety of delusions mention will be made of a few that have come under recent notice: "A young man of twenty believes that he is President; another patient, a driver, believed for ten months that he was the owner of a thousand horses, any one of which was worth thousands of dollars; he made a perfect recovery and now laughs at his old delusions. A young man of twenty-five believes his mother is not his mother, but the woman with whom he boarded, and that his brothers and sisters are her children but no relation to him. A young woman of thirty believes she is pregnant by a prominent merchant; the fact being she is not and never has been pregnant." The majority of the delusions are of an egotistic character, but lack the conduct or appearance of the position due to the character of the delusion. A patient with ragged clothing will assure you that he is worth millions, and yet sees nothing inconsistent between his delusions and his personal appearance. Another will assure you of his vast business interests, and yet remains contented in the hospital wards, laboring faithfully in the kitchen or laundry. A woman assures you that she is the great Patti, receiving thousands of dollars for each operatic performance, and yet is apparently happy in the sewing-room.

An *hallucination* is an imperfect perception through any one of the senses. A person who imagines that he sees something, or hears something, or tastes something, or feels or smells something that he is not seeing, hearing, tasting, feeling, or smelling, has an hallucination.

Delusional insanity is often based upon the development of hallucinations of the special senses, that of hearing being the most frequent; patients hear "voices" telling them what to do or not to do, and a delusion is built up and developed. Again, "voices" upbraid them or charge them with various acts, and upon this is developed a persecutory delusion that causes them much unrest. The following case has lasted for five years, and while the patient is at times apprehensive of some evil that may result to her, and uses judgment to protect herself, yet is not, nor never has been, melancholic, or shown any evidences, other than her present belief, of mental failure. She enjoys fair health, and partakes of the world's pleasures. Six years ago her husband suddenly died and the settling of a large estate was thrown upon the patient. Sitting in her hotel at the window, about five years ago, she saw a man come to the window in a building opposite to where she was, and make some motion to her. She was greatly alarmed. That evening, while walking on one of the busiest streets of the city, she distinctly heard a young man, in passing, make an improper proposal to her, and she has never walked on that street since without the same thing occurring, although not always by the same person. Her daughter, who accompanied her, did not hear the proposal, nor has she ever heard it, although, I regret to say, is gradually becoming convinced it must be true. Now for the sequel: the woman is not depressed or worried, shows no evidences of melancholia, talks about the affair as if it were a fact, which it unfortunately appears to her, and avoids the unpleasantness by never again walking on the particular street nor going in that neighborhood.

Again, visions appear, which result in delusions of personal importance. Taste and smell may be perverted, causing prolonged fasting, often from fear of poisoning.

Diagnosis.—*Delusional mania* and *delusional melancholia* are confounded with delusional insanity, the points of distinction being the absence of severe maniacal and melancholic acts; the patient simply possesses his insane delusion and may never refer to it unless questioned.

Paranoia or *monomania* and delusional insanity have many symptoms in common, but in the former "their whole thoughts and lives show a strong self-consciousness, and their egotism is intense" (Chapin); and if the patient believes he is Christ, he wishes to be so respected, and considers himself wronged if not so treated, while the delusional patient will say he is Christ and immediately drop the subject. There are, however, many borderland cases in which the diagnosis is difficult.

The distinction made here between paranoia and primary delusional insanity is not generally accepted.

Prognosis.—In acute primary delusional insanity, recovery is frequent, although the delusions may exist for a number of years. Many patients who make a complete recovery will still believe that their delusions were facts. A fair proportion of cases pass into the condition of chronic delusional insanity, this attack being the first or second period of Magnan's classification, viz.: *First period,* incubation or anxiety; *second period,* persecution; *third period,* grandeur, or ambition, or optimism; *fourth period,* dementia.

Treatment.—A supportive plan of treatment, with thorough action upon the bowels, kidneys, and skin, and plenty of fresh air, is of great value in all cases of delusional insanity. If the disease is the result of excesses, a course of strychnin and arsenic is indicated. A tranquil condition of the brain is essential, and few combinations are so valuable as digitalis and hyoscin, in small, repeated doses. Insomnia is an annoying symptom in many cases, and is best overcome by a digestible meal at bed-time, or a warm or hot bath in the evening, and if these fail a full dose of somnal, well-diluted, or trional, gr. xxx (2 Gm.), an hour before bed-time, in milk or spirits should be administered.

The following is of value:

℞. Somnal	ʒ iii	12 Cc.
Glycerin	℥ ss	16 Cc.
Tr. cardamom. comp. ...	℥ ss	16 Cc.
Aq. menth. pip. ..q. s. ad	℥ iii	90 Cc.

M. S.—Half tablespoonful, repeated in two hours.

PARANOIA.

Synonyms.—Monomania; chronic delusional insanity: reasoning mania; Verrücktheit.

Definition.—A chronic mental disease characterized by fixed logic or systematized delusions of persecution, of unseen or impossible agencies, or of self-exaltation, the emotions and memory being only paroxysmally defective, while the life of the individual is dominated by the delusions.

The term paranoia is now commonly used to cover a group of insanities which are degenerative in origin, chronic in course, and characterized by systematized delusions, with little impairment of the emotional faculties, and is not generally accepted as a synonym for monomania.

"A primary developmental insanity." "A condition of degeneracy." "There is imbecility of mind and there are delusions" (Dewey).

Causes.—There is generally a hereditary predisposition to insanity in monomania or paranoia. The exciting cause may be the result of an acute mania or melancholia, or the result of alcoholism, or of malnutrition in those who have had a struggle to keep their position in the world. Extreme worry in individuals with mental instability is a common cause. It may follow primary or acute delusional insanity.

Symptoms.—The course of monomania is essentially chronic, the delusions becoming fixed upon one particular subject, or set of subjects, which in turn dominate the life of the individual. The most common characters of these systematized delusions are delusions of persecution or suspicion, delusions of exaltation or of pride, and delusions of unseen agents or influences.

Delusion of exaltation or pride is well shown in the case of a woman of average talents and education, who had devoted much time, thought, and worry to a number of worthless patients, and now that she is in an insane asylum believes she has been placed there that others may reap the rewards of her inventive genius; she is constantly annoyed by what the physicians, attendants, and patients are doing, claiming that many such acts are for the purpose of annoying or harming her, her suspicions being of the most aggravating character.

Delusion of exaltation or pride is also well shown in the case of a man who believes he is Jesus Christ, and is angered almost to the point of homicide if great consideration is not shown him. Another male, whose origin is from the lower walks, believes he is to marry a distinguished authoress, and will resent any doubt of his intentions with blows.

Delusion of unseen agencies is well shown in the case of a female, aged forty years, who labored under the delusion that she was beset by numerous devils in her abdomen, the real cause being the presence of a cancer of the liver. Patients of this character often complain of electric influences, telephonic communications, and invisible agents tormenting them.

The range the delusions of monomania assume, is most wide and varied, but always associated with the *ego*. The patient is being persecuted not because, as in melancholia, he has committed some sin, or thinks he has, and deserves punishment, but because the persecutors wish to deprive him of his rights, titles, or estate, or degrade him or in some way injure him.

Diagnosis.—In the diagnosis of monomania there are three points to ever keep in mind: *first,* the duration—the fixed, systematized delusions must have existed over one year; *second,* the absence of symptoms of mania or melancholia; and *third,* the presence of systematized delusions affecting the personnel of the individual.

Prognosis.—Monomania is an incurable disease. Unless tuberculosis develop within a few years, dementia results.

Treatment.—The various methods adopted for building up and maintaining the tone of the body are applicable in this affection. The symptoms should be combatted as they arise on general therapeutic principles.

GENERAL PARALYSIS.

Synonyms.—General paresis; general paralysis of the insane; paresis; paretic dementia.

Definition.—A subacute or chronic, degenerative disease of the brain, sometimes involving the spinal cord, characterized by alterations in the intellectual and moral character, with the development of unsystematized ideas of self-importance or delusions of grandeur, finally merging into dementia (preceded by either a mania or a melancholia), and the gradual development of tremor, slurring speech, pupillary changes, ataxia, trophic changes, and finally general paresis.

Causes.—General paralysis of the insane occurs chiefly between thirty and fifty-five years of age, and in the male more frequently than in the female, although a notable increase in the lower class of females is being observed. It usually affects the robust, middle-aged individual, rapidly destroying all intelligence and judgment, leaving him to exist, often for months, as a demented human automaton. General paresis is increasing, and someone has said its increase is in proportion to "syphilization and civilization."

Predisposing causes: Heredity; an ambitious overstraining for prominence, learning, or wealth; forced intellectual activity in those with imperfect or improper early training; or in those with an imperfectly developed or organized cortex; cranial injuries; and atheroma.

Exciting causes: Alcoholic and sexual excesses; syphilis; mental and physical overstrain; and worry. "In many cases I think the middle-aged general paralytic is suffering for the sins of his youth" (Clouston).

"General paralysis is not a penalty of high cerebral development, but the expression of a discrepancy—an inadequacy of some brains to sustain the strain to which the race, as a whole, is subjected" (Spitzka).

Pathologic Anatomy.—A condensed description of the pathologic basis of general paralysis is difficult. It may be described as a chronic, diffuse, cortical encephalitis. The microscopic changes in the cortex, according to Mendel, as quoted by Folsom, are as follows:

1. Increase of nuclei and new cell-formation are observed, some nuclei small, some large, and with such varying reactions to coloring agents as to suggest dissimilarity of origin. The stellate or "spider" cells are increased in the upper layer of the cortex, where some may be normally found, and extend to lower layers, as is not the case in normal brains; they, too, may be several times the usual size and also push through the white substance to the ependyma of the ventricles. Proliferation of neuroglia or connective-tissue, and in time sclerosis of the cortex, which involves the medullary substance also in a greater or less degree is common.

2. The larger blood-vessels may or may not be atheromatous; in the capillaries there is an increase of nuclei in the walls, with thickening and hyaloid degeneration.

3. In the nerve-cells, the ganglion-cells, there are granular and fatty degeneration of protoplasm, sclerosis, and atrophy.

4. Atrophy and final disappearance of the nerve-fibers is observed, not limited to the cortex. This condition is found in other brain diseases also—senile dementia and epilepsy, for instance.

5. Focal lesions of the most various kinds, degenerative changes in the spinal cord, the several forms of sclerosis, and myelitis are encountered.

The spinal cord undergoes atrophy with gray degeneration in posterior and postero-median columns, and in the posterior spinal nerve-roots.

Symptoms.—For clinical convenience the disease is divided into *three stages* — prodromal, maniacal, rarely melancholic, and the stage of dementia — although there is seldom a marked division between the stages.

The prodromal stage may exist unrecognized for months or longer. It begins by an alteration in the habits and character of the individual, such as spells of irritability and obstinacy, which will not admit of contradiction or opposition, and there is a general feeling of elation and *bien-être,* or egotism, shown by the exalted opinion of his own attainments and importance, and a great laudation of members of his family. He becomes boastful, untruthful, dishonest, and forgetful, neglecting engagements, business, self, and family. He frequently makes extravagant purchases and may waste large sums of money before his condition of irresponsibility is recognized, or may unwittingly resort to dishonest means to obtain money, as was shown in the case of an intelligent gentleman, who had squandered considerable money in unprofitable property, going to a railroad ticket-office, asking for a ticket, remarking he was without cash, writing a check for one hundred dollars on a bank he had never had an account with, receiving ninety-nine dollars in change, immediately going to a jewelry store and purchasing a lady's gold watch and chain, paying sixty dollars for the same, and then going to a pawnbroker's and pledging the watch and chain for forty dollars, and the following day going to the same ticket-office and buying another ticket, such as he had purchased with the fraudulent check, and on being arrested protested he had done nothing dishonest.

In many instances the patient develops ideas of an enterprising character, and resorts to all forms of expedients, which, to his mind, are going to improve his or his family's station and wordly condition. He determines to change his occupation or business, or attempts to instruct the authorities in what he conceives should be their duties.

Moral lapses of paretics are most frequent during this stage, consisting of acts of theft, drunkenness, violent impulses, or indecent assaults, in individuals who have previously possessed a good moral character. They become profane and vulgar, and often resort to sexual excesses. Associated with any of the above symptoms may be any one or more of the following physical conditions: tremor of the muscles about the mouth, naso-labial folds, and of the tongue, causing a slight slur or hesitating speech; alterations in the pupils, or one pupil becoming somewhat larger than the other, or the pupils may be contracted to pin-head size with loss of accommodation; attacks of vertigo, or epileptiform or apoplectiform seizures. The gastric, intestinal, hepatic, and nephritic secretions are disturbed, and there may be headache and insomnia. After a variable duration, continuing in a mild degree for many months, the second stage begins.

Second or *maniacal stage* is much the same as a severe attack of acute mania (megalomania), plus the physical signs of paresis and the delusions or ideas of grandeur. The patient is excessively restless, boasting of his great wealth, intentions, prospects, and influence; one moment the most important of individuals, the next giving away thousands, and, if doubt is expressed as to his ability to do so, making it millions and often billions; presenting houses and lands, titles and offices, with unstinted liberality. It is to be noted that these so-called delusions of the paretic are in reality conceptions, or an expansive delirium, for when contradicted the patient makes no effort to defend them; they seem to be really assertions and reassertions, continuing until incoherency restrains the airy imagination. If questioned as to his health, replies, enthusiastically, "First-rate; never better in my life." The patient is sleepless, noisy, and destructive, with attacks of blind, uncalculating violence, resisting all who attempt to restrain or molest him. The violent impulses of paretics are similar to the furious excitement of the post-epileptic maniac.

The physical signs are more pronounced: the characteristic hesitating and slurring speech increases; the pupillary changes become more marked; the tremor of the tongue and lips increasing and spreading to the upper extremities; the gait ataxic; the patellar reflex increased, or, rarely, diminished; the sphincter of the bladder disordered, and sometimes there occurs paralysis of the anal sphincters.

During the progress of the second stage are developed cerebral crises,—syncope, petit or grand mal, apoplectiform attacks, or paralytic seizures. Few cases but show one or more of these conditions. There also occur myosis and loss of light reaction, and increased wrist and elbow jerks. The maniacal stage is of shorter duration than any other, and is usually succeeded by the—

Stage of dementia, the patient presenting all the evidences of failing mentality, with paralysis, trophic changes, as shown by the occurrence of bed-sores, cystitis, diarrhea, and arthropathies, or Charcot's joints, the patient emaciating rapidly, death closing the scene within a few months. Rarely the maniacal stage is preceded or replaced by a condition of melancholia with expansive hypochondriacal delusions. In a few instances a genuine lucid interval has followed either the prodromal or maniacal stage.

The *spinal form* of general paresis is fairly frequent, in which symptoms of spinal sclerosis are added to the mental ataxic phenomena of the usual form.

"Of the many divisions of general paralysis into several clinical types, all of them naturally more or less arbitrary, I know no other so satisfactory as Meynert's eight" (Folsom):

1. Simple progressive dementia, with the usual motor impairment which accompanies it, but, excepting hypochondrical depression, not necessarily exhibiting other mental symptoms than dementia.

2. With the expansive delusions and the distinctive motor disturbances, which appear simultaneously and are progressive, constituting the "classic" form of general paralysis. The mental state is usually self-satisfaction and exaltation, but there may be depression.

3. Of the same type as the last, but failing its steadily progressive character through arrest of the active process. The remissions, which seldom last so long as a year, raise hopes of recovery, but still manifest unmistakable impairment of the reasoning faculties. The psychic disturbances are much greater than can be accounted for by the atrophy of the brain alone.

4. Cases in which the characteristic exaltation and grand delusions reach such an astonishing height that manifest motor symp-

toms are looked for with confidence from day to day, and yet may not appear even for a year, any slight incoördination naturally being obscured by the general muscular disturbance. Meanwhile there may be such an improvement that the patient leaves the hospital for a while, once, rarely twice, on the responsibility of his family, but to return with marked motor as well as mental signs.

5. A very rare form, with alternate symptoms of exaltation and depression of the type of circular insanity.

6. With early furious delirium, painful hallucinations, confusion and incoherence somewhat resembling acute delirium.

7. Progressive general paralysis, in which the characteristic indications appear secondary to other forms of insanity; for instance, after paranoia or melancholia, first described by Hoestermann.

8. The combined form with sclerosis in the whole cerebrospinal tract, the symptoms of tabes or spastic paralysis predominating, according as the posterior or lateral columns of the spinal cord are chiefly involved. The ascending type, in which the cord is first affected, is rare. Optic neuritis, ending in atrophy and paralysis, especially of the ocular muscles, may precede marked mental symptoms.

Diagnosis.—The development of the following symptoms removes all difficulties in diagnosis: *Mental*—alteration in character, loss of memory, defective will-power, changed moral sense, insomnia, violent impulses, melancholia or mania, unsystematized delusions of expansive character, with an exalted sense of well-being, gradually ending in dementia. *Physical*—hesitating, slurring speech; tremor of the lips, tongue, and upper extremities; pupillary changes, myosis, loss of light reaction; exaggerated wrist-, elbow-, and knee-jerk; attacks of syncope, vertigo, epileptiform seizures, ataxia, trophic changes, and finally paralysis.

Paralytic insanity, organic dementia, or dementia paralytica, is not the same condition as general paralysis. It is the form of mental failure succeeding to gross brain lesions, such as apoplexy, tumors, softening, trauma, and sclerosis, associated with either hemiplegia or paraplegia.

Prognosis.—Unfavorable. Remissions very rarely occur. The duration of general paresis has been considerably lengthened by the hospital care of such patients now in vogue in all properly conducted institutions for the insane.

Treatment.—The care of the general health and meeting symptoms as they arise is all that can be done for general paresis. It is claimed that if the condition be recognized early in the prodromal stage, the stage of cerebral congestion or vasomotor paresis, much good may be accomplished, and, if not cured, may be held in check for a long period of time by the use of such drugs as digitalis or ergot.

The maniacal excitement may be quieted by the use of the hot bath, isolation (not seclusion), and the administration of small doses of hyoscin hydrobromate, which seems to exert an alterative action on the brain. For the insomnia, trional, gr. xx-xxx (1.3-2 Gm.), repeated, is usually satisfactory.

If a reliable syphilitic history is obtained, a thorough course of mercury and iodids should be administered. All means that promote the constructive metamorphosis are indicated in this most characteristic, progressive malady.

DEMENTIA.

Synonym.—Acquired feeble-mindedness.

Definition.—A progressive general weakening of the mind, characterized by a loss of reasoning capacity, a diminution of feeling, a weakened volitional and inhibitory power, and failure of memory, associated with lack of the power of attention, interest, and curiosity, in varying degrees, in an individual previously possessed of these mental qualities.

Forms.—*Acute dementia; alcoholic dementia; dementia apoplectica or paralytica; dementia choreica; chronic or secondary dementia; dementia epileptica; organic dementia; partial dementia; primary dementia; dementia senilis; dementia syphilitica; dementia toxica.*

Causes.—Deficient or feeble mental inheritance; age; atheroma; mania, melancholia, paranoia, and other forms of insanity; organic brain conditions; alcoholism; syphilis; developmental changes; climacteric.

Pathology.—In *acute dementia* the changes are dynamic. In the *primary dementia* there is probably atrophy of certain cells from over-stimulation, the tissues being normally deficient. In *secondary dementia* the chief changes are: "alteration in the size of the vessels, owing to the thickening and distention, the thickening being most marked in the deep layers, and in the walls of the vessels are fatty granules and hematoidin. The perivascular canals are enlarged. The changes in the cells may be described as deficiency in the number of pyramidal cells, and a want of distinctness of outline and branches, the nuclei being larger, but changed in form, and only capable of slight carmine staining." In *senile dementia* there is general atrophy and degeneration of all the tissues of the brain.

Symptoms.—The onset, extent, and variety of the impaired mentality differ greatly. In some patients the evidences of the failing mind are seen with the subsidence of the mania, melancholia, or other insanity, or soon after the development of the particular cause, while in another group of cases the development is slow and insidious. The difference in the intensity is marked; in one case the changes being scarcely noticeable, the patient being simply less active than before, showing a slight indifference to his environment; while in others the patients remain for hours alone, making no effort at movement and with little or no expression of the face; while still another class of cases are oblivious to the demands for food or drink, or the calls of nature, existing "in the darkness of perpetual intellectual and moral night." Between these symptoms are all varieties and degrees of mental enfeeblement, the physical symptoms of dementia varying with the particular cases, many enjoying the best of health, eating and sleeping well; while others are always unwell, first one organ and then another being affected; still another group suffer from chronic diarrhea, which finally causes death. Dementia patients seem predisposed to tuberculosis, nephritis, and epilepsy.

Acute dementia, or "stupor with dementia," is to be distinguished from "stupor with melancholia." The onset is rather sudden, with or without mania or melancholia, after some brain or bodily exhaustion, shock, or fright; the patient, a young person, "is horror-stricken, paralyzed in mind, not merely deranged, not depressed or excited, but deprived of feeling and intellect; his movements, if there be any, are automatic, but frequently he is motionless, standing or sitting, staring at vacancy for hours and days" (Blandford). These patients will not converse, and do not reply to questions, or but slowly, and in monosyllables, and their faces have a blank expression. One young man of thirty-three years, but three years in America, having an excellent musical education and remarkable skill as a piano performer, being unable to secure pupils to instruct, was obliged to accept a position as a piano-player at a questionable summer-resort garden, where he contracted the alcoholic and sexual habit. His excesses increased, although never intoxicated; he suddenly developed symptoms of dementia, his mind becoming a complete blank, his circulation feeble, the surface cold; and he never offered to enter the dining-room, and yet attended to the calls of nature. He never spoke, and would remain alone and motionless for hours. The sweetest music caused no movement showing intelligence. He was placed on the Mitchell rest treatment for six weeks, and, as his bodily condition improved, he was daily taken to the piano, and his fingers made to touch the keys. For weeks he showed no interest, when, slowly, one day he feebly ran his fingers over the keys, and from that day improved, until, within four weeks, his performance on the piano attracted wide attention, and, after recovery, which was complete, with no recollection of this sickness, he secured pupils and is to-day a successful teacher. He has assured me that he suffered no pain, or depression, but that all is a blank to him.

Alcoholic dementia, the mental weakness resulting from excessive use of alcohol. *Inebriety* is a form of dementia, there existing an uncontrollable alcoholic habit, with weakened or absent will power and impaired mentality. Sutherland defines seven forms of insanity from alcoholic excess: (1) Intoxication; (2) delirium tremens; (3) mania-a-potu; (4) dipsomania; (5) mania of suspicion; (6) chronic alcoholism or dementia; (7) general paralysis.

Dementia apoplectica or *paralytica* is an organic or terminal dementia due to the cerebral changes sometimes following a severe apoplectic seizure, and is usually associated with hemiplegia.

Dementia choreica is a feeble-mindedness associated with chronic or hereditary chorea, or, in some cases, probably the result of chorea.

Chronic dementia is the designation applied to all forms of dementia that have existed for one or more years.

Dementia epileptica is the slow mental impairment resulting from long-continued and frequently occuring epileptic convulsions.

Organic dementia, the mental deterioration resulting from gross organic brain lesions, such as sclerosis, tumor, embolism, or trauma. As an example of this form, the following case may be cited: An intelligent machinist, aged forty years, fell a distance of twenty feet, striking on his head, but without causing any fracture. He was unconscious one week, and on slowly recovering it was noticed that there was some change of character, which has grown most decided, and is associated with persistent insomnia. He is restless, indifferent; has loss of memory, is vulgar and profane, and inclined to be talkative—opposite traits to his former self; has violent outbreaks, and has a delusion that he is to make a fortune out of a polish the formula for which was given him by God, but which he has mislaid. He cannot read or write, or, at least, he will never make the attempt. His physical condition is good.

Partial dementia is an incomplete form of dementia in which the mental enfeeblement is associated with such a degree of intelligence and memory that the qualifying term "partial" is applicable. This variety of dementia constitutes the majority of able-bodied, working, chronic insane patients seen in insane hospitals.

Primary dementia is seen most frequently in the young, developing slowly and insidiously, without any symptoms of mania or melancholia, usually in a youth who has given promise of a bright future, by a slowly progressive indifference to his former occupation, studies, or surroundings, with developing carelessness and negligence of person and proprieties, no amount of external stimulus serving to rouse the receding mentality, until finally the downward course ends in dementia so decided that, but for the history of the individual, the case would be classed as congenital, or imbecility.

Secondary, sequential, or *chronic dementia,* is the most common variety of mental impairment following mania, melancholia, and other insanities. According to Bevan Lewis, twenty percent of manias and fifteen percent of melancholias become permanent dements.

Dementia senilis is the result of cerebral atrophy, with its consequent failing mental power. Loss of memory for recent events is one of the most common symptoms. The disease often begins as a senile mania, melancholia, or delusional insanity. The following is an average case: A female aged sixty years, with intemperate history, was, on admission, exceedingly filthy and covered with many vermin. She says she has been persecuted in her poverty; that she could not obtain goods from the store when she had no money, though the shopkeeper was rich; that she was neglected by others; insists that she ought to have been assisted; is unconcerned with her surroundings; is trifling and disrespectful, restless, moving her hands and body almost continually; is childish and silly in manner, frequently laughing, claiming she is happy, and will not work; cannot remember her only sister's name or where she herself last resided.

Dementia syphilitica is the feeble-mindedness resulting from cerebral syphilis. This group of patients is always sanguine, and assert they are "all right," "never sick in my life," and yet unable to assist or care for themselves. This form of dementia has many symptoms akin to general paresis, and, indeed, is often termed "pseudo-paresis."

Dementia toxica is the mental failure produced by the long-continued and excessive use of opium, cocaine, and chloral. Chronic plumbism is also given as a cause.

Diagnosis.—Acute dementia is often misnamed melancholia with stupor, but if the patient is in the teens the probabilities are that it is a case of the former, while if past forty it is almost certainly the latter.

The distinction between dementia and idiocy or imbecility must always be determined. Esquirol's graphic description is well worth remembering: "The dement was a rich man who has become poor; the idiot, on the contrary, has always been in a state of want and misery."

Prognosis.—Acute dementia is generally favorable. All other varieties are incurable. The average life-time of dements is placed at about twelve years, the great majority dying of tuberculosis, nephritis, or apoplexy.

Treatment.—Patients suffering from acute dementia should be placed on the Mitchell rest régime, with attention to all the seerations. If Dr. Mitchell's directions are carefully followed, the great majority of cases of acute dementia will recover within nine to twelve months.

For the other forms of dementia, unfortunately, there is no cure, the treatment resolving itself into attention to the general health, with proper custodial oversight.

DISEASES OF THE SKIN.

General Symptomatology.—To acquire even the most slight knowledge of diseases of the skin, a definite understanding of the individual lesions, or objective phenomena, must be obtained, as it is the aggregation of these lesions that constitutes the external manifestations of these affections and the basis of diagnosis. Some lesions are initial manifestations and are termed *primary lesions*; others result from various modifications of the original lesions and are termed secondary lesions.

The primary lesions are macules, papules, vesicles, blebs, pustules, tubercles, wheals, and tumors. These represent definite structural changes in the skin. The definitions of these terms by various dermatologists must therefore be very similar.

Macules are variously sized and shaped discolored areas of the skin characterized by the absence of elevation or depression.

Papules are circumscribed solid areas of elevation of the skin, the size of which varies from that of a pin-head to a pea.

Vesicles are circumscribed elevated areas of the skin containing clear or opaque fluid, varying in size from a pin-head to a pea.

Blebs, or bullæ, are round or irregularly-shaped epidermal elevations containing clear or opaque fluid and varying in size from a pea to a goose-egg.

Pustules are circumscribed epidermal elevations containing pus, and varying in size from a pin-head to a finger-nail.

Wheals, or pomphi, are circumscribed, edematous elevations of the skin, of a fugitive, or ephemeral, character.

Tubercles are circumscribed, solid, pea-sized elevations, situated deeply in the skin.

Tumors are variously sized, shaped, and constituted prominences, the seat of which is in the deep layers of the integument.

The *secondary lesions* include scales, crusts, excoriations, fissures, ulcers, scars, stains, and any other secondary structural change. These manifestations do not bear directly on the diagnosis and their detailed description may therefore be omitted.

Subjective symptoms in dermatologic affections are itching, burning, tingling, smarting, pain, and sense of heat. The intensity of these manifestations is necessarily subject to great variation. They may exist separately or in different degrees of combination.

ANEMIA OF THE SKIN.

Anemia of the skin consists in a diminution in the quantity or alteration in quality of its blood-supply and may be general or local, transient or persistent. Generalized dermal anemia occurs as a part of a general anemia from various causes, and as a result of cerebral anemia. Localized anemia follows the application of cold, pernio, frost-bite, Raynaud's disease, emboli and thrombi, keloid, morphea, scleroderma, alopecia areata, cicatrices, etc. Transient anemia occurs as the result of shock, syncope, anger, fear, hemorrhages, etc. Persistent anemia is that which occurs as the result of some structural change in the skin such as morphea and alopecia areata; it also accompanies persistent general anemias.

HYPEREMIA OF THE SKIN.

Hyperemia or congestion of the skin consists in an increase of its blood-supply due to overfilling of the blood-vessels without other structural change. As in other parts of the body, it may be active or passive, idiopathic or symptomatic. The most important hyperemias are erythema simplex and erythema intertrigo.

Erythemia simplex is a circulatory disturbance of the integument characterized by variously sized and shaped areas of redness unattended by elevation or depression. It is accompanied by mild itching or burning, and disappears on pressure. It may be due to exposure to extremes of temperature (*erythema caloricum*), exposure to the sun's rays (*erythema solare*), injury (*erythema traumaticum*), irritation of poisonous plants (*erythema venenata*), or to the absorption of drugs, antitoxins, ptomains, etc., (*toxic erythema*).

The treatment should be internal and external. As many of these cases are due to intestinal autointoxication, fractional doses of calomel followed by a saline purgative should be administered routinely, except in those instances in which the affection is obviously due to external causes. Externally, dusting powders such as zinc oxid and starch, and sedative lotions such as saturated boric acid solution are very beneficial.

Erythema intertrigo or chafing, is the variety of hyperemia encountered in regions such as the buttocks, genital regions, and flexures of joints, in which the skin surfaces are in apposition and rub one on the other. In the obese and in infants it is very frequent and often develops into a true dermal inflammation. Redness, heat, and burning are the only symptoms. Cleanliness and the local application of dusting powders such as magnesium carbonate, zinc oxid, subnitrate of bismuth, talc, etc., serve to prevent and relieve the condition.

INFLAMMATIONS OF THE SKIN.

ERYTHEMA MULTIFORME.

Erythema multiforme is an inflammatory disease of the skin characterized by symmetric, bright or dark reddish, more or less variegated macules, papules, and vesicles, occurring discretely or in patches, often sharply defined, and marginate, of various size and shape, running an acute course (Duhring). Constitutional disturbance usually precedes or accompanies it and is manifested by feverishness, malaise, rheumatoid pains, anorexia, etc. This is followed by the sudden appearance of the eruption which may consist of macules, papules, vesicles, or blebs, the eruption being designated by the predominant type of lesions. It is bright or dusky red in color at first, but soon becomes purplish or bluish and shows a great predilection for extensor surfaces such as the backs of the hands and dorsal surfaces of the feet. Other portions of the body, however, are not exempt. The subjective symptoms are itchng and burning.

The direct cause of the affection is undetermined but early adult life, spring and autumn seasons, changes in the weather, and the rheumatic diathesis are known to influence its production materially. The pathologic changes consist in dilatation of the dermal vessels with moderate serous and cellular exudation into the tissues. In distinguishing the condition from other cutaneous affections it should be remembered that the eruption appears suddenly in crops lasting from one to four weeks, is multiform in character, is most marked on extensor surfaces, has a purplish red or violaceous color, is accompanied by constitutional symptoms, and undergoes spontaneous involution often reappearing when the necessary contributory conditions (such as change in the weather) are supplied.

Treatment.—In all cases, the administration of fractional doses of calomel followed by a saline is of benefit. Quinin and the salicylates often serve to hasten the eruption's involution and to relieve the constitutional manifestations. Locally, antipruritic lotions are of value. The following may be used:

℞.	Acid carbol.	ʒ ii	8. Cc.
	Glycerin	ʒ ii	8. Cc.
	Aquæ	O i	480.0 Cc.

M. S.—Poison, apply locally twice daily.

ERYTHEMA SCARLATINOIDES.

Erythema scarlatinoides is a variety of exudative erythema resembling scarlatina in its cutaneous manifestations but differing from it in its other characteristics. The eruption appears suddenly with very slight constitutional reaction, is punctiform or diffuse, and disappears by desquamation in from one to six days. The face is seldom attacked; the strawberry tongue is absent; recurrence is common; and the affection is non-contagious. It may arise as an idiopathic condition or it may accompany septicemia, pyemia, ptomain poisoning, rheumatism, uremia, and the infectious fevers. A similar eruption may follow the absorption of quinin, salicylates, copaiba, belladonna, and similar drugs. The affection is devoid of danger and terminates favorably usually within a week.

Treatment.—When the cause can be ascertained, it should be promptly removed in order to prevent persistence or recurrence of the condition. Usually no local treatment is required. Dusting powders or sedative lotions may be necessary if there is any attendant itching.

ERYTHEMA NODOSUM.

Erythema nodosum (*dermatitis contusiformis*) is an inflammatory disease attended by the formation of symmetric, round or oval, node-like swellings and accompanied by more or less constitutional disturbance. The onset is marked by slight fever, rheumatoid pains, and loss of appetite. These are shortly followed by the appearance of tense, rosy, red nodes or swellings, usually over both tibiæ, resembling erysipelas. They are at first hard and extremely tender to the touch but later soften and their color becomes that of a bruise. Suppuration never occurs and the lesions undergo spontaneous involution in from a week to ten days. The affection is usually observed in children and young adults particularly in the spring and fall. It is often associated with rheumatism and gastrointestinal disorders. The structural changes incident to this disease are congestion, serous and cellular infiltration, and hemorrhages. It is closely related to erythema multiforme.

Treatment.—Rest, with elevation of the affected parts is essential. The application of lead-water and laudanum serves to relieve the pain. On no account should the lesions be incised. Internally, fractional doses of calomel followed by a saline should be given to relieve the gastrointestinal tract of any offending material and to promote elimination. Quinin, salol, salicin, sodium salicylate, and phenacetin may be employed with benefit.

ERYTHEMA INDURATUM.

Erythema induratum (erythème induré des scrofuleux—Bazin) is an uncommon inflammatory affection observed in scrofulous individuals, particularly strumous girls, characterized by the formation of circumscribed infiltrated areas, usually in the calves of the legs, which terminate either in absorption or necrosis with the formation of an indolent ulcer. It occurs usually in winter in poorly nourished individuals with feeble circulation, and is extremely chronic with a tendency to recurrence.

Treatment.—In all cases it is extremely necessary first to improve the patient's general condition by good food, fresh air, sunshine, cod-liver oil, syrup of the iodid of iron, and similar measures. Locally, elevation of the parts is of decided advantage and in the event of ulceration, surgical cleanliness is all that is necessary.

URTICARIA.

Synonyms.—Hives; nettle-rash.

Definition.—An inflammation of the skin characterized by the development of wheals of a whitish, pinkish, or reddish color, accompanied by stinging, pricking, and tingling sensations, often associated with febrile and gastric symptoms.

Causes.—It is usually due to some indiscretion in diet. Certain substances such as fish, crabs, lobsters, cheese, sausage, buckwheat, strawberries, nuts, pork, etc., in susceptible individuals bring about an attack. Antitoxin serums, copaiba, quinin, cubebs, chloral, salicylic acid, morphin, etc., may also give rise to the condition. Intestinal parasites and undigested food are also causes. Locally, the bites or stings of insects, exposure to heat, the sting of the nettle, and traumatism such as caused by the stroke of a whiplash, often produce the condition. Less frequent causes include reflex irritation from hepatic, renal, uterine, or bladder derangements, puncture of pleural effusion, rupture of an hydatid cyst, malaria, emotion, neurotic conditions, purpura, pregnancy, lactation, and the menopause.

Pathology.—An acute edematous condition of the papillary layer of the skin, characterized by the rapid development of a "wheal"—a more or less firm elevation—consisting of a circumscribed collection of the semifluid material, the result of a rapid exudation into the upper layers of the skin. The production of the wheal is the immediate result of a disturbance of the vasomotor system, which is shown by the interference of the circulation in the wheal, the blood being driven from its center to its periphery, causing the whitish apex and red areola so characteristic of the developed "hive."

Symptoms.—An attack of "hives" is characterized by the sudden development of *wheals* upon the cutaneous surface, which usually as suddenly disappear, their site being temporarily marked by a spot of redness or hyperemia. With the appearance of the wheal occur distressing itching, burning, tingling, crawling, pricking, and stinging sensations, to relieve which the patient still further irritates, tears, or otherwise wounds the surface by scratching, whence are often developed deep-colored, flat, lenticular papules.

Very frequently an attack of "hives" is associated with fever, headache, and gastric disorder. The wheals may appear upon any portion of the body; their size varies from that of a pea to that of a walnut or an egg—"giant wheals;" the number varying, sometimes being so numerous as to cover the whole surface. The shape, size, color, and number of the wheals that may occur have given rise to a number of names to designate the lesions. Thus, *urticaria annularis* occurs in rings; *urticaria figurata* occurs in spirals; *urticaria vesiculosa* has a vesicular development on the summit of the wheal; *urticaria bullosa,* a bullous development at the summit; *urticaria papulosa,* or *lichen urticarus,* the wheal and a small papule are combined; *urticaria tuberosa,* or giant wheals; *urticaria hemorrhagica,* or *purpurata urticaria,* a combination of uticaria and purpura; *urticaria evanida,* a rapid appearance and disappearance of the lesion; *urticaria perstans,* slow disappearance; *urticaria conferta,* when the wheals are confluent; *urticaria pigmentosa,* when the wheals are succeeded by pigmentations of the site, the tints varying from dark brown, greenish yellow, to a chocolate color; *urticaria febrilis,* when the wheals are associated with fever; *urticaria ab ingestis,* when associated with indigestion; *urticaria factitia,* when the wheals are produced artifically.

Prognosis.—Acute attacks respond quickly to treatment, but recurrences are common.

Treatment.—In the early stage, an emetic will be of value but usually the condition is well-advanced when seen by the physician necessitating the administration of a brisk purgative. Following this some intestinal antiseptic such as salol, or sodium salicylate should be given. The diet should be as plain as possible, care being taken to eliminate those substances for which the patient has an idiosyncrasy. Among other drugs of value in this condition may be mentioned quinin, phenacetin, antipyrin, pilocarpin, atropin, tincture of belladonna, ammonium chlorid, arsenic and potassium bromid. The following pill is useful in many cases:

℞.	Pulv. pilocarpi,		
	Ext. guaiaci	āā gr. iss	āā 0.1 Gm.
	Lithii benzoat.	gr. iij	0.2 Gm.

M. S.—Two to four each twenty-four hours.

If there be atonic dyspepsia and constipation, the following combination is beneficial:

℞.	Magnesii sulphat.	℥ j	32. Gm.
	Ferri sulphat.	gr. xvj	1. Gm.
	Sodii chloridi	ʒ ss	2. Gm.
	Acidi sulphurici dil.	fʒ ij	8. Cc.
	Infus. cascarillæ	f℥ iv	120. Cc.

M. S.—Tablespoonful before breakfast, diluted.

Locally, baths, lotions, or dusting powders will be necessary to relieve the itching. Among the most serviceable measures are: sponging with alcohol, brandy, whiskey, carbolized water, or witch hazel, alkaline baths, and acid bath. Duhring recommends the following:

℞.	Acidi carbolici	ʒ iss	6. Gm.
	Glycerini	fʒ ij	8. Cc.
	Alcoholis	f℥ viij	240. Cc.
	Aq. amygdal. amar.	f℥ viij	240. Cc.

M. S.—Use as a lotion two or three times daily.

Bulkley suggests the following:

℞. Chloralis,
Camphoræ ....āā ʒ j   āā   4. Gm.
Misce, and incorporate
with Pulveris amyli ...℥ j-ij   32.-63. Gm.
M.—Keep tightly corked in a wide-mouthed bottle.
S.—Rub in with hand.

A serviceable formula is the following:

℞. Chloroformi ....fʒ j   4. Cc.
Ung. zinci oxid. ....℥ ij   63. Gm.
M. S.—Apply locally.

Or—

℞. Menthol ....gr. v   0.32 Gm.
Petrolat. ....℥ i   32. Gm.
M. S.—Apply locally.

Or—

℞. Acid Benzoic. ....gr. x   0.6 Gm.
Alcohol ....f℥ i   30. Cc.
M. S.—Apply locally.

Urticaria pigmentosa, already referred to, begins in the early months of infancy and is characterized by buff-colored wheals, with or without itching, that persist for a long period and after disappearing leave behind brownish stains. It is very rare. It is essentially chronic but seldom lasts until puberty. The treatment consists of internal and local medication based on the same principles employed in other varieties of urticaria.

ANGIONEUROTIC EDEMA.

Angioneurotic edema is a neurotic condition in which transient circumscribed, edematous swellings appear on the skin, and sometimes on the mucous membranes, and disappear after a variable period without leaving behind any structural alterations. It arises usually without obvious cause and is in all probability a vasomotor neurosis. Certain drugs in susceptible individuals may induce it. Recurrences are frequent, and when the larynx is involved the affection assumes a grave aspect. The treatment is similar to that of urticaria.

ECZEMA.

Synonyms.—Tetter; salt rheum; scall.

Definition.—A non-contagious inflammation of the skin, characterized by any or all of the results of inflammation, at once or in succession, such as erythema, papules, vesicles or pustules, accompanied by more or less infiltration and itching, terminating in a serous discharge, with the formation of crusts, or in desquamation.

Forms.—*Acute; subacute; chronic.*

Varieties.—*Eczema erythematosum; eczema papulosum; eczema vesiculosum; eczema pustulosum; eczema rubrum; eczema squamosum; eczema fissum; eczema verrucosum; eczema sclerosum.*

Causes.—Eczema attacks persons in all spheres—the rich, the poor, the infant or the aged, and males or females. Many families, especially those having the "catarrhal predisposition or peculiarity of constitution," seem more liable; indeed, it appears probable that a predisposition to eczema may be transmitted from parent to child. Other causes are: improper food, gastrointestinal disorders, imperfect elimination of products of waste, intestinal parasites, dentition, deficient urinary secretion, Bright's disease, diabetes, functional and organic nerve affections, the rheumatic and gouty diatheses, vaccination, prolonged contact of hot fomentations, contact with the poison vine (rhus toxicodendron) and poison tree (rhus venenata), heat and cold, and various chemic and mechanic irritants.

Pathology.—Eczema is a catarrhal inflammation of the skin—a dermatitis, with superficial serous exudation. There is first hyperemia, or congestion of the vessels of the skin. The hyperemia is soon followed by a serous exudation. If the superficial exudation be profuse enough to form small drops, and if the epidermis possess sufficient resisting power not to give way immediately before it, vesicles form, producing the variety known as eczema vesiculosum; if the vesicles contain a large admixture of young cells, so that the serum be turbid, yellow, and purulent, the vesicles become pustules, termed eczema pustulosum; if the serous exudation be not sufficient to either elevate or break through the epidermis, instead of either vesicles or pustules forming, there occur dry scales, rising from the reddened skin—eczema squamosum. When the exudation is sufficient to detach the epidermis, thus exposing the red and moist corium, it is termed eczema rubrum.

In chronic eczema, the skin is subacutely inflamed and is very much thickened, hardened, and infiltrated with cells which extend throughout the entire corium, even into the subcutaneous connective-tissue. The papillæ are enlarged and at times may be distinguished with the naked eye. Pigmentation may take place in the deep layers of the rete and in the corium, especially about the vessels.

Symptoms.—Eczema is the most common of all cutaneous affections, with symptoms varying in accordance with the particular variety of the affection and the location, although the general characteristics of a catarrhal inflammation are present in all; these are *redness,* either limited or diffused; *heat,* of the part affected; *swelling,* the result of the serous exudation, giving rise either to a *discharge* (weeping), with subsequent *crusting,* or to the deposition of plastic material. The most constant, annoying, and troublesome symptom is the *itching,* or, at times, *burning,* which varies from that which is simply annoying to that which is almost unendurable.

Eczema runs its course either as an acute affection, lasting a few weeks, not to return, or to return acutely at wide intervals, or, as is much more frequently the case, it assumes a chronic state, continuing with more or less variations for months, years, or even a life-time. It may appear upon any portion of the body or involve the whole integument (*eczema universale*). The varieties are named in the order the lesions assume at their commencement.

Eczema Erythematosum.—An erythema or redness of the surface, with a yellow tinge. The size of the macule may be very small or quite extensive, with irregular outlines. There may be slight swelling of the patch, but no discharge occurs unless it be where two surfaces come into contact (*eczema intertrigo*), as about the genitalia. Cases without discharge are covered after a few days with a thin film of dry, exfoliating epidermis or scale (*eczema squamosum*). When a discharge (weeping) or moisture occurs, it is followed with more or less crusting. Intense itching is a constant symptom. The variety occurs most frequently on the face, the back of the neck, and the genitalia.

Eczema Papulosum, or Lichen Simplex.—This variety of eczema appears in the form of small, rounded papules, the size of a pin-head, of bright-red or, at times, dark-red color; they may be either discrete or confluent. In some cases all, while in others a greater or less number, of the papules pass into vesicles and run much the same course as vesicular eczema. The itching is of the most intense character, leading to severe scratching, by which the summits of the papules are torn, causing them to bleed, the blood forming dark-red crusts. The arms and legs are most often involved.

Eczema Vesiculosum.—This variety begins with burning, pain, redness, and swelling, followed by the eruption of an immense number of minute vesicles, either discrete or confluent, rapidly distending with a clear or yellowish fluid and attended with intense itching. Soon the vesicles rupture, the fluid rapidly diffusing over the surface and drying into yellowish, honey-like crusts. New crops of vesicles soon follow, or if subsequent vesications do not occur, the fluid rapidly diffuses over the excoriated surface, which also, in turn, dries into large, yellowish crusts. After a variable time the various symptoms gradually subside. Intense itching is the most prominent subjective symptom, and gives rise to an irresistible desire to scratch. All portions of the body are liable to this variety of eczema, the most frequent location, however, being the face, and when occurring in this region in children is commonly known as *crusta lactea.* The affection is very chronic and recurrences are common. It often terminates in eczema rubrum.

Eczema Pustulosum, or Eczema Impetiginosum.—This form usually begins as vesicular eczema, the fluid rapidly changing to pus. After a short period, during which the pustules have increased in size, they burst and the escaped fluid forms thick, greenish-yellow crusts, which, in turn, rapidly dry and fall off, or crumble away. The location of this variety is most usually upon the scalp and face. It is observed most often in poorly nourished and unclean children and is stubborn to treatment. Itching is a prominent symptom.

Eczema Rubrum.—This is a variety only from a clinical standpoint. It may result from any of the foregoing varieties. The surface of the skin is inflamed and infiltrated, red, moist, and weeping, the profuse serum rapidly drying into thick, yellowish, greenish, or brownish crusts, the color depending upon the character of the fluid, which may be serum, pus, or blood from the exposed and lacerated corium. The crusts adhere closely and firmly to the part, and unless removed by mechanical means may remain indefinitely, the disease pursuing its course beneath. *Eczema rubrum*, or *madidans*, "presents two appearances—as it occurs with its crust, and as it exists without this covering. In the one case the skin itself is altogether obscured by a dirty, yellowish, or brownish crust; in the other the skin presents a bright or violaceous red, punctate, wounded surface, deprived in great part of its epidermis, and exuding a scanty or profuse, clear or opaque, syrupy, yellowish fluid. Sometimes this is streaked with blood." The itching and burning are severe. It may develop upon any portion of the body, but is most commonly seen upon the legs, particularly in elderly people, and on the face in infants. Its course is chronic and tends to increase in severity.

Eczema Squamosum.—This is also a clinical variety. It occurs as the terminal stage of the erythematous, vesicular, pustular, or papular varieties of the affection, but more particularly the first named. A typical case presents itself in the form of variously sized and shaped reddish patches, which are dry, or more or less scaly, the skin being more or less infiltrated or thickened. When occurring at the flexures of the body the skin is liable to become fissured. Its course is usually chronic.

Eczema Fissum, or Rimosum.—A clinical variety, in which, during the progress of the erythmatous, vesicular, or pustular varieties of eczema, cracks or fissures result when the lesion occurs upon regions subject to constant motion, such as between the fingers, toes, nates, and the various joints. At times the fissures are extensive and deep, and of a bright-red color, showing the true skin, and intensely painful upon motion. Chapped hands are typical instances of fissured eczema.

Eczema Sclerosum.—This clinical variety of eczema, occuring most commonly on the palms, soles, and finger-tips, is characterized by hypertrophy of the papillæ, showing itself as hard, thickened, infiltrated, localized patches, which are very liable to crack (eczema fissum).

Eczema Verrucosum, or Papillomatosum, differs from the foregoing in that the thickened, infiltrated patch has a warty, verrucous appearance. Its course is chronic.

Acute and Chronic Eczema.—The line which divides these two conditions is drawn by means of the clinical and pathological features. The course of eczema, in the majority of instances, is chronic. It may be said that so long as the general inflammatory symptoms are high and the secondary changes slight, the affection is acute, and that when the process has settled itself into a definite line of action, continually repeating itself and accompanied by secondary changes, it is chronic.

Diagnosis.—The many varieties in which eczema manifests itself renders the diagnosis a matter of importance. The following characteristic features of eczema are of value in arriving at a diagnosis: Inflammation, redness, swelling, edema, thickening from cell infiltration, serous exudation followed by crusting, on the removal of which a moist surface is exposed, absence of a sharp line of demarkation between the diseased area and the healthy skin, polymorphism of the lesions, and marked itching and burning.

Erysipelas may be confounded with erythematous or vesicular eczema. The points of difference are the fever and other general disturbances. The deep-seated inflammation of the skin, rapidly spreading, with heat, swelling, and edema without moisture, giving the surface a deep-red, shining, and tense appearance, are characteristic of erysipelas and serve to distinguish it from eczema.

Herpes zoster may be confused with vesicular eczema but in the former the eruption is preceded by neuralgic pains over the affected areas, vesicles form in groups along the course of superficial nerves and are large and tense, showing no tendency to spontaneous rupture, the eruption is unilateral, and the course is definite; all of which features are absent in vesicular eczema.

Scabies often resembles eczema, especially those cases in which the resulting dermatitis is severe. A distinction here is of the utmost importance as the apparent eczema may persist indefinitely unless the true cause be promptly ascertained. In scabies the eruption is distributed to the flexor surfaces of the body, the webs of the fingers, axillæ, mammary glands, buttocks, penis and inside surfaces of the legs and thighs; the face is exempt except in nursing infants; the itching is worse at night; there is a history of contagion; and the burrows and itch-mite may be detected. Eczema has no characteristic distribution, is not contagious; the itching is constant, and there are no burrows or itch-mites.

Impetigo contagiosa may be at times mistaken for pustular eczema, the points of distinction being: In impetigo there is a history of contagion; the lesions first appear as discrete vesicles or blebs, the contents of which rapidly become purulent and soon flat, loosely-attached crusts form. The lesions are superficial and disappear within one or two weeks. Itching is very slight. In eczema there is a diffuse deep inflammatory base; the affection is non-contagious, and the course very chronic.

Sycosis vulgaris may be distinguished from eczema by its localization to the hair-follicles, the interfollicular skin being free, and by its great tendency to recur.

Psoriasis may be readily differentiated from eczema by its location (elbows, knees, scalp), the presence of round, sharply marginated patches abundantly covered with imbricated, silvery, mother-of-pearl scales, the absence of infiltration, thickening, and discharge so characteristic of eczema, and the very chronic course.

Tinea circinata and squamous eczema may simulate each other. The former is characterized by its circinate sharply-defined patches, clearing in the center and spreading on the periphery, its contagious nature, and the presence of the tricophyton fungus in the scales, demonstrable by the microscope. In eczema, the patches are irregular, not well-defined, and do not tend to clear in the center. It is not contagious and there is no fungus present in the scales.

Seborrhea of the scalp and squamous eczema of the same region closely resemble each other. In eczema, however, the skin is more or less red, inflamed, and thickened, and the scales larger, less abundant and less greasy and drier than seborrhea. In eczema the scales are usually seated in a circumscribed patch, while in seborrhea, as a rule, they cover the scalp uniformly. Itching occurs with both disorders. The history of the two affections should be of material aid in rendering the diagnosis clear; still, however, in many cases the diagnosis is difficult. Both are frequent affections.

Treatment.—There is no specific. The indications are to remove the cause if possible, to maintain the general health, and to apply such substances to the diseased area as will induce involution of the inflammatory process. The diet should be given most careful attention as frequently, particularly in children, errors in this direction are most potent etiologic factors. Tea, coffee, alcoholic beverages, sugar, candies, pastries, starchy foods, fried meats, condiments, etc., should be interdicted or at least reduced to a minimum. Substances which the patient is aware do not agree with him should of course be avoided. Fresh air and moderate exercise are essentials in the treatment together with attention to the secretions, particularly of the kidneys. Sluggish action of the bowels should be avoided by the use of such mineral-spring waters as the Hathorn, or Hunyadi Janos, or a morning dose of magnesium sulphate. The "acid mixture of iron" is of great value in this connection:

℞.	Ferri sulphatis	gr. viii	.51 Gm.
	Magnesii sulphatis	℥ iss	47. Gm.
	Acid sulphuric. dilut.	♏ lxxx	5. Cc.
	Tr. Cardamom. comp.	q. s. ad f℥ iv	120. Cc.

M. S.—One tablespoonful in water, half an hour before breakfast.

For children, equal parts of aromatic syrup of rhubarb and castor oil make an excellent laxative mixture. The addition of magnesia to syrup of rhubarb may also be employed. Calomel and soda is a useful combination.

If the urinary secretion be small and the urine heavy, full doses of potassium acetate and large draughts of water should be used. If either a rheumatic or gouty tendency exists, the salicylates, lithium salts, or colchicum should be employed. If there is any scrofulous or tubercular tendency cod-liver oil and syrup of the iodid of iron should be prescribed in addition to fresh air, sunlight, regulated exercise, etc. In anemic individuals, iron, quinin, strychnin, mineral acids, syrup of the hypophosphites, and small doses of corrosive sublimate are indicated. *Arsenic is usu-*

ally contraindicated in eczema, its field of usefulness being limited to those cases occurring in weak, anemic, debilitated, neurotic individuals. It has no direct bearing upon the disease itself. Potassium iodid, internally, frequently aids in reducing the infiltration in chronic thickened eczemas even in the absence of syphilis.

Locally, the first step in the treatment is to remove all forms of existing irritation. It may be stated as a principle, that nothing irritant is ever to be applied to the surface in acute eczema and that in chronic eczema, stimulation is indicated. Soap and water are to be avoided on areas the seat of acute eczema. For cleansing purposes, water containing boric acid, bran, starch, or oatmeal, may be employed. In chronic, thickened, and indurated eczema, soap is often of value as a stimulant. Crusts and scales are nearly always present in eczema and must first be removed to obtain the best results from local applications. This may be readily accomplished by saturation with oily preparations, a starch or other mild poultice, a saturated solution of boric acid, or dilute boroglycerin. Pastes and ointments may be easily removed by first softening with olive oil, sweet oil, or petrolatum. Soap and water should not be used for either purpose.

Acute eczema. If the type of disease is vesicular, dusting powders such as magnesium carbonate, boric acid, bismuth subnitrate, starch, zinc oxid, talcum, etc., may be employed with advantage in the early stage. The following may also be used:

℞. Pulv. camphoræ ....ʒ j 4. Gm.
Zinci oleat. ....ʒ ij 8. Gm.
Pulv. amyli ....℥ j 32. Gm.
M. S.—Dusting powder.

J. C. White recommends bathing the part with *lotio nigra* full strength or diluted with lime-water, applied by means of a sponge or a piece of cloth for ten or fifteen minutes at a time, and at intervals of a few hours or longer, the sediment being allowed to dry on the skin, after which oxid of zinc ointment should be gently rubbed over the part. As a rule, the itching and burning are promptly relieved and the affection often arrested.

℞. Hydrarg. chlorid. mit. ...gr. viii 0.5 Gm.
Liquor calcis ....f℥ i 30. Cc.
M. S.—Lotio Nigra. Apply locally as directed.

Stelwagon employs the boric acid lotion, 15 gr. (1 Gm.) to the ounce (30. Cc.), followed by applications of zinc oxid ointment in vesicular eczema and the following compound lotion in erythematous and papular types:

℞.	Acid. boric ....ʒ ii	8. Gm.
	Acid. carbolici ....ʒ ss	2. Gm.
	Glycerin. ....♏ x-xxx	0.65-2. Cc.
	Aquæ ....q. s. ad O ss	250. Cc.

M. S.—Apply locally twice daily.

This may be used alone or followed by an ointment or a dusting powder. Lotions containing an excess of the substance in solution are of value by the sediment they leave behind, which acts as a dusting powder. The calamin and zinc oxid lotion may be taken as an example:

℞.	Calamin,	
	Zinc oxid. ....āā ʒ ii-iv	8.-16. Gm.
	Liquor calcis ....f℥ ii	60. Cc.
	Aquæ vel solut. acid. boric saturat. q. s. ad O ss	250. Cc.

M. S.—Apply locally several times daily. (Stelwagon).

The following lotion is employed extensively in acute cases by Hartzell:

℞.	Resorcin ....ʒ ss	2. Gm.
	Bismuth subnitrat. ....ʒ ii	8. Gm.
	Glycerin. ....ʒ ii	8. Cc.
	Liq. calcis .....q. s. ad f℥ iv	120. Cc.

M. S.—Apply locally twice daily.

Some cases do better on ointments such as—

℞.	Zinci oleat. ....ʒ iv	16. Gm.
	Olei olivæ ....fʒ iv	16. Cc.

M. Ft. unguentum.

S.—Apply locally twice daily.

Or, bismuth oleate, made according to the following formula of McCall Anderson:

℞.	Bismuthi oxidi ....ʒ j	4. Gm.
	Acidi oleici ....℥ j	30. Gm.
	Ceræ albæ ....ʒ iij	12. Gm.
	Vaselini ....ʒ ix	36. Gm.
	Ol, rosæ ....♏ ij	0.12 Cc.

M. S.—Use locally as directed.

If the discharge be excessive, the following formula of Bartholow is valuable:

℞. Plumbi acetat.	℥ ss	16. Gm.
Pulv. camphoræ	gr. xv	1. Gm.
Ol. amygdal.	f℥ ij	60. Cc.
Cerat. flav.	℥ j	32. Gm.

M. S.—For local application.

The late Dr. Frank Maury was partial to the following formula in vesicular eczema:

℞. Hydrargyri chlor. mitis	gr. xx	1.3 Gm.
Ung. zinci oxid. benz.	℥ j	32. Gm.

M.—Et ft. unguentum.
S.—Apply twice daily.

Pastes are often of value, of which Lassar's paste may be taken as a type:

℞. Pulv. amyli,		
Pulv. zinci oxidi	āā ʒ ii	8. Gm.
Petrolat.	℥ ss	16. Gm.

M. S.—Apply to affected area twice daily; using sweet oil or petrolatum to remove the same before applying fresh paste.

This may be used alone or in combination with boric acid or salicylic acid or it may be rendered more soft by the addition of an equal quantity of petrolatum. Diachylon ointment, made by the formula of Hebra or by melting four parts of diachylon plaster and two or three parts of olive oil, is also very efficacious.

In pustular eczema, the following is of great benefit:

℞. Hydrarg. ammoniat.	gr. xx	1.3 Gm.
Petrolat.	℥ i	32. Gm.

M. S.—Apply locally.

For eczema papulosum the following lotions are particularly valuable:

℞. Acidi carbolici	ʒ j-ij	4.-8. Gm.
Glycerini	fʒ iv	16. Cc.
Alcoholis	fʒ iv-vj	16.-24. Cc.
Aquæ destil.	ad Oj	ad 480. Cc.

M. S.—Apply locally. (Duhring).

Or—

℞.	Thymol	gr. xv	1. Gm.
	Alcoholis	f℥ j	30. Cc.
	Aquæ destil.	f℥ j	30. Cc.

M. S.—Apply locally.

To relieve the itching incident to acute eczema, carbolic acid, menthol, and preparations of tar may be added to the preceding formulas but in very weak strength in order to avoid inducing additional irritation.

After the disappearance of the acute symptoms, the applications should be more stimulating and should include carbolic acid, thymol, tar, oil of cade, and similar substances. It is to be remembered, however, that the more chronic the affection and the less violent the inflammatory symptoms, the more successful is tar in this disease. Furthermore, it should be borne in mind that the dosage of external medication is subject to great variations in different individuals and it is always best to begin with a very weak ointment or lotion, watching the effects while the strength is being increased. Unless this precaution is taken the disease is likely to be aggravated. The following is of value in subacute cases:

℞.	Liq. carbonis detergens. .	℥ ss-ii	16.-64. Cc.
	Aquæ	O i	480. Cc.

M. S.—Poison; for external use only.

Or—

℞.	Liq. plumbi subacetat. . .	fʒ ss	2. Cc.
	Tr. picis mineralis comp.	fʒ ss	2. Cc.
	Aquæ destillat.	f℥ viii	240. Cc.

M. S.—Use freely as a lotion. (Hutchinson).

Duhring considers the following one of the most elegant of the tar ointments:

℞.	Olei cadini	fʒ iss	6. Cc.
	Cerati simplicis	℥ j	32. Gm.
	Ol. amygdal. amar.	gtt. x	0.6 Cc.

M. Ft. ungt.
S.—Apply as directed.

Or—

℞.	Picis liquidæ	fʒ j	4. Cc.
	Glycerini	fʒ j	4. Cc.
	Alcoholis	fʒ vj	24. Cc.
	Ol. amygdal, amar.	gtt. xv	1. Cc.

M. S.— To be rubbed firmly into the skin.

The following is Bulkley's valuable "liquor picis alkalinus":

℞.	Picis liquidæ	fʒ ij	8. Cc.
	Potassæ causticæ	ʒ j	4. Gm.
	Aquæ destillatæ	fʒ v	20. Cc.

The potassa to be dissolved in water and gradually added to the tar with rubbing in a mortar.

M. S.—To be used diluted.

Chronic Eczema. In this variety the treatment varies according as to whether the affected area is weeping or is dry, hard, and sclerosed. The weeping and discharging eczemas while chronic as regards time are usually acute in type and require sedative or moderately stimulating applications until the discharge has been reduced. In the dry, infiltrated, and thickened varieties, stimulation is necessary. The following is of value:

℞.	Olei cadini	ʒ i	4. Gm.
	Acid. salicyl.	gr. x	0.65 Gm.
	Pulv. amyli,		
	Pulv. zinci oxidi	āā ʒ ii	8. Gm.
	Petrolat.	ʒ iv	16. Gm.

M. S.—Apply locally twice daily.

The following treatment, first suggested by Hebra, is of great benefit in the treatment of chronic eczema of the legs:

"A lump of green soap (made originally of herring-fat and potassa, and containing three percent of caustic potassa), the size of a small nut, is smeared upon a piece of wet flannel and applied to the affected part, and firmly rubbed until the soap has disappeared, when the flannel is to be dipped into warm water and again applied to the part and rubbed until an abundant lather forms, more water being added from time to time until the suds are most abundant, when the surface is thoroughly washed and freed from all the soap and carefully dried, after which the following (Hebra's diachylon) ointment, having been spread before the application of the soap, is to be applied. It is prepared as follows:

"Fifteen ounces of the best olive oil are added to two pounds of water, and heated to boiling in the water-bath. Three ounces and six drams of an equally good article of litharge (plumbi oxidum) are dusted over the fluid in ebullition, which is constantly stirred throughout, in order to prevent the formation of fatty acids. During the cooking, water is occasionally added as required. The stirring is to be continued until the ointment is quite cold.

"The ointment is spread upon strips of soft muslin and the affected part enveloped, care being exercised that neither folds nor wrinkles occur, the whole being covered by a firm roller and the patient being able to go about as usual. The entire operation is to be repeated twice daily."

A modification of the above ointment, technically known as "*unguentum diachyli albi of Hebra,*" has been successful in a number of cases. The formula is—

℞. Emplast. plumbi,
Vaselini ....āā ℥ j   āā 32. Gm.
Ol. lavandulæ ....q. s.   q. s.
Dissolve with heat and stir till cold.
M. S.—Apply on strips.

The application of an elastic webbing bandage (not the ordinary rubber bandage) to the affected leg in cases of eczema unattended by discharge is often of great benefit. The support afforded by a gelatin dressing commends its use but it is contraindicated when there is any active discharge. The following is used extensively:

℞. Zinc. oxid,
Gelatin. ....āā 30 parts.
Glycerin .... 50 parts.
Aquæ .... 90 parts.

M. S.—The application is melted on a water-bath and the temperature is allowed to fall until it is near that of the body, after which it is thickly painted on the already cleaned affected area. It is then strewn with a powder or absorbent wool dabbed on to facilitate the hardening process. (Whitfield).

Da Costa claims to have had excellent results in the treatment of eczema rubrum from the internal administration of the solution of arsenic and the iodid of mercury (Donovan's solution), ♏ii-v (0.12-0.3 Cc.), in water after meals and the local application of the following:

℞.	Ung. plumbi subacet. ....ʒ iv	16. Gm.
	Acid. carbolici cryst. ...gr. iij	0.2 Gm.
	Petrolat. ....ʒ iv	16. Gm.

M. S.—Apply freely on muslin strips.

TREATMENT OF SPECIAL FORMS AND VARIETIES OF ECZEMA.

The principles upon which eczema is treated admit of no variation, no matter in what region the disease is encountered, but the form of irritaion to which the affection is usually due, differs in different parts of the body and requires more than passing mention.

Universal eczema arises either as the result of grave internal disorders, especially in children, or from generalized irritation such as accompanies the parasitic affections and attends certain occupations. Obviously the removal of the cause in these cases brings about subsidence of the eczema.

Eczema capitis is either erythematous, vesicular, or pustular in character. If the first named, it at once tends to become chronic, settling into the variety known as eczema squamosum, often involving the entire scalp and accompanied with intense itching. The pustular variety is the more common form, occurring upon the scalp of children and young adults, existing as a few patches, or, what is more frequent, involving the entire scalp. The pustules soon rupture, the liquid drying into greenish-yellow crusts, often covering the whole scalp with a cap of crust. The hair becomes matted and caked, the sebaceous secretions collect, and if the part is not cleansed, becomes offensive. In severe cases of pustular eczema of the scalp enlargement of the lymphatic glands of the back of the neck and of those behind the ear occur; but they never suppurate. Pediculi are frequently associated with eczema capitis in children, either as a primary cause or a result of the matted condition of the hair which constitutes a favorable habitat for them. Care should always be taken not to confuse eczema of the scalp with psoriasis, seborrhea, syphilis, tinea favosa, and tinea tonsurans.

Treatment.—In the pustular variety the crusts should first be removed by saturation with olive oil or oil of sweet almond and washing with warm water and soap, or the use of a starch poultice or a twenty-five percent solution of boroglycerin. It should be borne in mind that neglect is responsible for a great many of these cases. Da Costa recommends the following application after removal of the crusts:

℞.	Hydrargyri chlor. mitis	gr. xx	1.3 Gm.
	Acid. carbol. cryst.	gr. iij	0.2 Gm.
	Petrolat.	℥ j	32. Gm.

M. S.—Apply thoroughly.

The late Ellerslie Wallace was fond of using the following:

℞.	Sodii carbonatis	gr. xxx	2. Gm.
	Petrolat.	℥ j	32. Gm.

M. S.—Apply thoroughly after removal of the crusts.

In cases associated with pediculi or succeeding impetigo contagiosa the following is of great benefit:

℞.	Hydrargyri ammoniat.	gr. x-xx	0.65-1.3 Gm.
	Adeps benzoat.	℥ j	32. Gm.

M. S.—Apply locally.

For the squamous variety of the scalp, the following formula, recommended by Duhring, is excellent:

℞.	Picis liquidæ	fʒ j	4. Cc.
	Glycerini	fʒ j	4. Cc.
	Alcoholis	fʒ vj	24. Cc.
	Ol. amygdalæ amar.	gtt. xv	1. Cc.

M. S.—Diluted or full strength, rubbed thoroughly into the scalp.

Other applications, such as boric acid lotion, oxid of zinc ointment, etc., previously advised, may also be used with the exception of the pastes, which are extremely difficult to remove from the hair. A word of caution may be given regarding resorcin lotions in eczema of the scalp. In brunettes such applications may be used, but in blondes the hair is apt to assume various shades as a result of the application.

In all cases cleanliness is essential. The diet should be carefully regulated and tonics should be administered if the patient's condition is one of general debility.

Eczema faciei. In this location the affection may be either acute or chronic. In adults the erythematous variety is frequently encountered in patches about the forehead and cheeks. It usually results from irritation such as accompanies exposure to heat and cold and contact with strong soaps, etc. Eczema of the face is more common in children, however, the varieties being the vesicular and pustular. It is seen on the forehead, nose, and upper lip, and is associated with severe itching. The primary cause in most cases is to be found in disturbances of digestion although the condition may be greatly aggravated by attempts at treatment by members of the family.

Treatment.—The cause should be promptly removed. The difficulty with which determination of the cause is attended should lead the physician to regulate the diet, as already given, and interdict the local use of soap and water routinely. The application of a mild sedative lotion such as a saturated solution of boric acid should be prescribed at first until the true condition of the disease is asserted. In erythematous eczema of the face lotions seem most beneficial; in vesicular and pustular forms, soft ointments and pastes are valuable. Late in all forms of facial eczema pastes are of great service. The following is also useful:

℞. Zinc. oleat. ....ʒ i 4. Gm.
Petrolat. ....℥ i 32. Gm.
M. S.—Apply locally.

Eczema labiorum. Eczema attacks the lips, either alone or in connection with other parts of the face. One or both lips may be affected. The irritation of the tooth brush or tooth powder may be the cause. The symptoms are swelling, redness, heat, infiltration, slight scaliness, and fissures. The affection may be in the skin around the border of the mouth, or the vermilion and mucous membrane of the lips. The mouth may be contracted and the lips partly glued together by the exudation and crusts. Eczema labiorum may be confounded with herpes labialis and syphilis.

Treatment.—This is very difficult and discomforting to the patient. Among the remedies at times successful may be mentioned silver nitrate, potassium nitrate, carbolic acid, boric acid, or tar in solution or ointment and flexible collodion. A combination of boric acid, acetanilid, and bismuth is often of value. Tragacanth, acacia, and gelatin paints are also used and should be applied with the lips apart, otherwise the dressing cracks when the mouth is open.

Eczema palpebrarum.—This is a rather common occurrence in scrofulous children, showing itself along the edge of the eyelids. It is frequently accompanied by conjunctivitis. It may be due to the congestion that attends eyestrain. Pediculosis ciliorum is an occasional cause. Pustules form at the openings of the hair follicles and the lids become crusted. Swelling, redness, and itching are present and unless the parts are frequently cleansed, the lids will become glued together.

Treatment.—The discharge incident to any existing conjunctivitis should be removed and any ocular condition present should be treated. Yellow oxid of mercury, gr. i (0.065 Gm.), in petrolatum, ʒ ii (8. Gm.), rubbed into the roots of the eyelashes every night is very beneficial. The oleate of zinc and the glycerite of tannic acid are also valuable.

In severe cases the plan recommended by McCall Anderson should be pursued. It consists in the extraction of the eyelashes and touching the edges of the lids with a solution of potassium in water, ten grains to the ounce. The edges should be carefully dried and the lid everted, a very small quantity on a delicate brush being applied, immediately neutralizing the alkali with acetic acid or vinegar.

Eczema narium. This also occurs most often in children and appears as the pustular form of the disease. Nasal catarrh and general malnutrition are the most important etiologic factors. These should first receive attention, after which the treatment advised for pustular eczema, elsewhere, is applicable.

Eczema barbæ.—Eczema of the beard is characterized by the formation of extensive pustules, showing a preference for the skin about the hairs, drying as yellowish or greenish crusts, matting the hairs together and adhering to the parts. The affection may be confined to the hairy portions of the face, or extend to other regions of the face, localized or general, acute or chronic.

Eczema barbæ in its general features somewhat resembles both tinea sycosis and sycosis non-parasitica, but sycosis is an inflammation of the hair-follicles only and is rarely associated with crusting, while crusting is abundant in eczema.

Treatment.—The hair should be kept very short by clipping with scissors; when the inflammation has subsided somewhat, shaving closely is indicated. Sedative lotions and ointments are to be used at first. The crusting should be removed by oil or petrolatum but not by soap and water, if the inflammation is acute. In chronic cases the following ointment should be applied after cleansing and shaving the beard:

℞. Hydrargyri ammoniat. ..	gr. xv-xxx	1.-2. Gm.
Sulphur. præcipitati	ʒ ss-j	2.-4. Gm.
Petrolat.	℥ j	32. Gm.

M. S.—To be thoroughly applied.

As in other forms of eczema, internal treatment may be of value. The solution of arsenic and iodid of iron (Donovan's solution), ♏ii-v (0.13-0.3 Cc.), three or four times daily, is often of benefit.

Eczema aurium.—Eczema of the ears may be either erythematous, vesicular, or pustular. If the former, thickening results, with desquamation of flakes or large scales; if either of the latter, crusts form, which may envelop the whole ear, the symptoms being swelling, redness, and severe burning and itching, and if the process extend into the meatus, occlusion may result, causing temporary deafness. The most characteristic symptom of erythematous eczema of the external auditory canal, besides the appearance of small flakes, is intense and persistent itching. It often results from the irritation of a discharge from the ear and from treatment directed toward the middle ear.

Treatment.—For acute vesicular or pustular eczema, removal of the crusts and the use of calomel as an ointment in the strength of thirty grains (two grams) to the ounce (thirty-two grams). If chronic, the use of tar, as already suggested. For chronic erythematous eczema of the external auditory canal, the following formula has generally controlled this stubborn condition:

℞. Hydrargyri flav. oxid. ...	gr. j-iij	0.065-0.2 Gm.
Morphinæ sulph.	gr. j	0.065 Gm.
Vaselini	ʒ ij	8. Gm.

M. S.—Apply to the canal.

Eczema genitalium.—This is a most distressing condition. In the male, the scrotum and penis are involved alone or together, the former alone being the more common, and is complicated with eczema of the inner side of the thigh or thighs. The symptoms are: swelling, often edema, moisture, crusts, and painful fissures, followed by extensive thickening accompanied with intense itching. In the female, the affection attacks the labiæ, and, rarely, the vagina and mons veneris, and may extend to the surrounding parts, especially to the perineum. The symptoms of eczema of the labia are: great swelling, edema, redness, with great heat and a free discharge, forming crusts, which are apt to glue the apposing surfaces together. If the variety be the erythematous, in place of a discharge with crusts, the symptoms named are followed by slight scales. The itching is most violent and distressing. Uncleanliness and neglect serve to aggravate the condition. Glycosuria may be a factor in its production.

Treatment.—The parts attacked should be kept constantly enveloped in cloths wet with a saturated solution of boric acid until the more pronounced inflammatory symptoms subside, when the boric acid may be used as a dusting powder, completely enveloping the parts. Mild solutions of menthol are valuable. Tincture of myrrh or witch hazel, well-diluted, are excellent applications. The following is an excellent application for eczema of the scrotum:

℞.	Acidi borici	q. s. for sat. sol.	
	Tinct. myrrh.	f℥ ss	16. Cc.
	Tinct. camphoræ	f℥ ij	60. Cc.
	Hydrarg chlor. cor.	gr. iij	0.2 Gm.
	Aquæ destil. ...	q. s. ad f℥ viij q. s. ad	240. Cc.

M. S.—Apply several times daily.

An excellent formula for eczema of the vulva is—

℞.	Iodoformi	ʒ ss	2. Gm.
	Bal. Peruviani	fʒ j	4. Cc.
	Petrolat.	℥ j	32. Gm.

M. S.—Apply on soft cloths.

Eczema of the genitalia is always obstinate to treatment and requires constant attention to both the local and general condition of the patient. Other remedies employed in eczema, elsewhere, should be given a fair trial.

[illegible]

[illegible] than the oper- [illegible] of lint or gauze.

[illegible] and more particularly those [illegible] of a vesicular eczema, with [illegible] and burning, and unless speedily relieved. [illegible] The pain on nursing becomes so severe [illegible] compelled to refuse the child. It must be borne [illegible] also occurs in women who are not [illegible] single women.

Treatment.—Tilbury Fox advises the following plan:

"1. Great cleanliness and care in washing away any remnants of milk after each time the child is put to the breast; and if the nipple be tender and excoriated, use—

"2. A little lead-water and calamin powder, as follows:

℞.	Liq. plumbi	fʒ iss	6. Cc.	
	Pulv. calaminæ præp.	ʒ iss	6. Gm.	
	Glycerini	fʒ j	4. Cc.	
	Adeps	℥ j	32. Gm.	M.

"3. I cover over the nipple with a lead nipple-shield. This excludes the air, keeps the part from being chafed, and I think the lead does good after the part has become less red and sore. I often use a little glycerite of tannic acid, painted on night and morning.

"The above application can always be removed with a little cold cream and a little warm-water sponging before the child goes to the breast."

Eczema palmarum et plantarum.—The features of the affection in both these regions are identical. The diagnosis is often obscured by the thickened state of the epidermis. The symptoms are, infiltration, thickening, callosity, moisture followed by dryness, and fissuring, the last named frequently becoming so deep and painful that the patient is unable to use his hands, or, if on the soles, to walk. The affection is always chronic, affecting either of the parts alone, or all at one and the same time. Itching is a constant and annoying symptom.

The diagnosis is to be made between eczema of these parts and psoriasis or syphilis.

Treatment.—The plan of Hebra for eczema rubrum will usually be successful for this variety. The following formula is also valuable:

℞.	Hydrargyri oleat. 5-15 per cent.	ʒ iv	16. Gm.
	Olei cadini	fʒ ss	2. Cc.
	Cerat. simplicis	ʒ iv	16. Gm.

M. S.—Rub well into part morning and night, first macerating with hot water.

Eczema unguium.—The nails are seldom attacked alone, but in connection with eczema manuum. The symptoms are roughness, want of polish, unevenness, and a punctate or honeycomb appearance, similar to that seen in psoriasis of the nails. The nail becomes depressed, particularly at its root, thus interfering with its nutrition, resulting in loss of this appendage.

Treatment.—Internally, arsenic is a valuable remedy. Locally, the following:

℞.	Ung. picis liq. ....ʒ iv	16. Gm.
	Hydrargyri chlor. mitis..ʒ ss	2. Gm.
	Vaselini ....ʒ iv	16. Gm.

M. S.—Apply thoroughly.

Eczema crurum.—Eczema of the legs is usually encountered in poor persons past middle age. Varicose veins are nearly always present. The treatment should be mild and soothing at first, including boric acid lotion or resorcin lotion through the day and a paste applied as a plaster. While the area is discharging it should not be bandaged. Whenever possible, rest should be procured. After the moist character has disappeared, the diseased area may be stimulated by the various means already mentioned and the leg supported by a bandage, preferably one of elastic webbing.

In all cases of eczema, the use of composite remedies of unknown proportions is dangerous, as in most cases the disease is aggravated. As previously stated, there is no specific for this affection; the applications and dosage varying with different individuals and with different types of the disease.

ECZEMA SEBORRHOICUM.

Seborrhoic eczema is an inflammatory disease beginning primarily on the scalp and extending to the face, chest, and elsewhere over the body, characterized by irregular patches of redness, or yellowish redness, and scaliness. It is probably due to a parasite. The scales are grayish or dirty-white in color and greasy to the touch. It resembles seborrhea but has an additional inflammatory element absent in that affection.

The treatment consists in removing the scales by means of soap and water, after which a weak sulphur ointment (gr. xxx to the ounce) or resorcin lotion (gr. viii to the ounce) should be applied. The scalp should receive careful attention.

IMPETIGO CONTAGIOSA.

Definition.—An acute contagious inflammatory disease characterized by the development of one or more discrete superficial vesicles or blebs, of various size and shape, the contents of which soon become purulent, producing yellowish or brownish crusts.

Causes.—The affection is observed most often in poor and unclean children. It is contagious and autoinoculable. Occasionally it follows vaccination. When occurring in the scalp, it may nearly always be traced to pediculosis capitis. Institutions, laundries, barber shops, etc., often aid in producing epidemics. The exciting cause is inoculation with the ordinary pus microörganisms.

Pathology.—Among the microörganisms capable of producing this affection may be mentioned the staphylococcus aureus, staphylococcus albus, streptococcus, and occasionally the tricophyton fungus. The bleb is formed between the rete mucosum and the horny layer of the epidermis, the roof-wall being afforded by the latter. A mild inflammatory reaction surrounds the lesion having its seat in the upper layer of the corium. The bleb contains a whitish-yellow fluid, pus corpuscles, blood corpuscles, epithelial cells, cellular detritus, and microörganisms.

Symptoms.—The eruption appears with greatest frequency on the face and hands and often some very trivial injury as a scratch. As the lesions appear, their contents are clear, but within a day or so the vesicle or bleb character is lost and they assume all the features of pustules. The pustules rapidly dry, forming crusts which have a " stuck on " appearance. After several days these drop off leaving a slightly red area beneath. Usually, there are present at the same time, several lesions in various stages of development. The vesicopustules may coalesce forming several large blebs or they may arrange themselves in half circles. Itching is very slight or absent. Neglect or improper treatment may cause the development of an eczema on an impetiginous area. The duration of the disease is in most instances from ten days to two weeks.

Treatment.—The crusts should be removed by means of some nonirritating soap and water after which the following may be applied:

℞. Hydrarg. ammoniat.	gr. v	.3 Gm.
Hydrarg. oxid. rub.	gr. v	.3 Gm.
Petrolat.	℥ i	32. Gm.

M. S.—Apply locally twice daily. (Stelwagon).

Other mild antiseptics such as boric acid, sulphur, etc., may be employed, care being taken to reduce the strength sufficiently to avoid irritation.

ECTHYMA.

Definition.—An affection of the skin, characterized by the formation of one or more large, isolated, flat pustules, situated upon an inflamed base.

Causes.—It is most common among those who live in squalor and poverty, in unclean adults, and in delicate and poorly-nourished children. Improper and insufficient diet, want of ventilation, excessive work, and uncleanliness are all prominent causes. It should be remembered that ecthymiform lesions also occur at times in the course of pediculosis, scabies, and syphilis.

Pathology.—The lesion is a typical pustular process, severe but superficial, and not extending beyond the papillary layer of the corium. The pustule is situated upon a firm and highly inflamed base; the number varies from one to a dozen or more. With the disappearance of the lesions pigmentation and scarring may follow. The exciting cause of the affection is the presence of pus-producing microörganisms in the debilitated skin.

Symptoms.—The disease is characterized by the development of one or more, round or oval, flat, pustules, about the size of a pea or bean, attended with moderate heat, burning, and pain, and, if the number be large, slight febrile reaction. The pustules are first yellowish in color, surrounded by a firm and sensitive bright-red areola, the pustule afterward becoming reddish from the admixture of blood, soon drying into flat crusts of a brownish color. The duration of each pustule is between two and three weeks, new ones forming until the cause is removed. The most prominent sites are the thighs, legs, shoulders, and back.

Diagnosis.—Ecthyma may be distinguished from other pustular affections by its predilection for the legs of unclean persons, usually adults, and by the presence of discrete, flat, deep-seated pustules with broad inflammatory areolas.

Treatment.—Without treatment the affection may persist indefinitely but under proper care the response is prompt. A bath with a change of clothing should begin the treatment. A search should be made for animal parasites and if found, appropriate measures should be instituted. Nutritious food and tonics should be given. Locally, cleanliness and the following application are of great value:

℞.	Acid carbol.	gr. v	.33 Gm.
	Hydrarg. ammoniat.	gr. xxx	2. Gm.
	Petrolat.	℥ i	32. Gm.

M. S.—Apply locally twice daily.

DERMATITIS HERPETIFORMIS.

Synonyms.—Duhring's disease; hydroa; herpes gestationis.

Definition.—An inflammatory, superficially seated, multiform, herpetiform disease, characterized mainly by erythematous, vesicular, pustular, and bullous lesions, occurring usually in varied combinations, accompanied by burning and itching, pursuing usually a chronic course, with a tendency to relapse and recur (Duhring). The affection occurs most often in middle life and arises from a number of causes, chief of which are disturbances of the nervous system.

Treatment.—The cause should be ascertained and removed. Tonics, especially arsenic, act most favorably on the disease. Rest, nutritious diet, and attention to personal hygiene are important. Phenacetin, acetanilid, belladonna, and cannabis indica will often relieve the symptoms when administered internally. Locally, applications containing carbolic acid, resorcin, tar, sulphur, and ichthyol are of value. Duhring recommends a strong sulphur ointment in the vesicular and pustular types.

PEMPHIGUS.

Definition.—An inflammatory disease of the skin, either acute or chronic, characterized by the development of a succession of rounded, irregularly-shaped blebs or bullæ, varying in size from a pea to an egg.

Causes.—Obscure. Nervous prostration, general debility, heredity, female sex, disorders of menstruation, pregnancy, etc., are important indirect etiologic factors.

Pathology.—The affection is considered to be a trophoneurosis. The blebs are situated in the epidermis and probably arises from a sudden effusion from the vessels of the corium as the result of dilatation. The contents of the blebs or bullæ are yellowish or colorless serum, of a neutral or alkaline reaction; the older the fluid, the more alkaline it becomes. In the late stages of a bleb the fluid becomes puriform. In rare instances blood is contained in the bleb (*pemphigus hemorrhagicus*). The papillary vessels are dilated and the papillæ, corium, and subcutaneous tissue are edematous and infiltrated with leukocytes.

Symptoms.—There are two varieties: *pemphigus vulgaris; pemphigus foliaceus.*

Pemphigus vulgaris may be acute or chronic and may or may not be accompanied by febrile reaction. It is manifested by the successive development of blebs varying in size from that of a pea to an egg, of a round or oval shape containing a colorless fluid, the color becoming yellowish or puriform as they grow older. They arise abruptly from the sound skin with a definite line of demarkation, unattended by symptoms of inflammation. A characteristic feature of the eruption is the successive appearance of the lesions; one crop no sooner disappears than another forms, each crop running its course in from three to six or ten days. The limbs, face and trunk are the regions most often affected but the condition may also involve the mucous membranes. Itching and burning of a mild degree are present; occasionally they are very severe (*pemphigus pruriginosus*).

Pemphigus malignus is characterized by the great size and number of the blebs, which coalesce, rupture, and are succeeded by excoriated surfaces, which occasionally taken on ulcerative action, seriously impairing the patient's health.

Pemphigus foliaceus differs from pemphigus vulgaris in that the blebs, instead of being distended or tense, are flaccid and only partially filled with fluid, and they rupture before arriving at their state of full development. This variety also appears and disappears in crops. After rupture the fluid immediately dries into thin, whitish flakes, which are detached in quantity, leaving a red, excoriated surface—the rete and corium. If the affection has continued for some time, the skin presents the appearance of a superficial scald. The course of this variety is esentially chronic. All portions of the body-surface are liable to the lesion, as is also the mucous membrane of the mouth and vagina. It is most common, however, upon the limbs.

Pemphigus vegetans is a rare variety of the disease in which wart-like vegetations develop upon the sites of the ruptured blebs.

Diagnosis.—The disease is rare, and the presence of blebs in any given case should direct the attention to pemphigus only after bullous erythema multiforme, impetigo contagiosa, bullous syphiloderm, and bullous eruptions of artificial production have been excluded. The characteristics of pemphigus are its chronicity, and the appearance in crops of large, tense, abruptly elevated non-inflammatory blebs.

Prognosis.—The outlook is uncertain. Most cases pursue a very chronic course ultimately ending in death from some intercurrent disease. Mild attacks are less liable to persist and often end in recovery. Constitutional disturbances, extensive involvement of the skin, and the presence of flaccid or hemorrhagic blebs are unfavorable signs.

Treatment.—The general health should be restored and maintained by rest, nutritious food, and the administration of tonics especially arsenic and quinin. Locally, the blebs should be punctured as soon as formed and dusting powders such as boric acid, zinc oxid, or starch should be applied locally. Sedative lotions are also valuable. Hebra advises the continuous bath.

POMPHOLYX.

Synonyms.—Dysidrosis; cheiro-pompholyx.

Definition.—An acute inflammatory disease of the skin, affecting especially the hands and feet, characterized by the appearance of vesicular, vesicobullous, and bullous lesions, attended by burning, tingling, or itching.

Causes.—It occurs in adults of both sexes, and is believed to depend upon some general disturbance of the nervous system. Mental overwork and lowered nerve-tone are ascribed as causes. The disease is considered to be a vasomotor neurosis.

Symptoms.—The lesions are distributed symmetrically, and occur as deep-seated tense vesicles usually on the lateral and palmar aspects of the hands, fingers, feet, and toes, accompanied by itching and burning. They may coalesce but more frequently they remain discrete, showing no tendency to rupture. The contents are absorbed and desquamation follows. The duration of the attack is from several days to a few weeks. Recurrence is common.

Diagnosis.—The affection may resemble vesicular eczema and dermatitis venenata. The distinctive features of pompholyx are the location, the tense, deep-seated character of the vesicles which do not tend to rupture, the subjective symptoms, the absence of acute inflammation, and the tendency to recur.

Prognosis.—The acute attacks seldom last more than one or two weeks and the course is seldom influenced by treatment. The tendency to relapses has already been mentioned.

Treatment.—Recurrence may be prevented to a great extent by the employment of measures calculated to improve the general health. Locally, pastes, ointments, or lotions containing antipruritic drugs often allay the subjective symptoms.

HERPES SIMPLEX.

Synonyms.—Fever blisters; "cold sore."

Definition.—An acute inflammation of the skin, characterized by the development of one or more groups of vesicles, filled with a clear serum, occurring for the most part about the face (*herpes facialis*) and genitalia (*herpes progenitalis.*)

Causes.—Herpes facialis occurs during the course of febrile and nervous disorders and is often associated with gastrointestinal disorders. Herpes progenitalis usually arises from uncleanliness and friction.

Pathology.—The affection is neurotic in origin, and by some observers believed to be due to a toxic neuritis of a cutaneous nerve.

Symptoms.—The appearance of the vesicles is usually preceded by a feeling of heat in the region, together with slight tumefaction or swelling. Rarely the herpetic attack is attended with malaise and pyrexia. The eruption usually appears in the form of a small cluster of pin-head to split-pea sized vesicles, containing a clear fluid, becoming cloudy, afterward puriform, and drying in small, yellowish or brownish crusts; they are few in number, and may coalesce. They disappear without leaving a scar.

Herpes facialis; occurs upon any portion of the face, but most frequently about the lips—*herpes labialis.* The alæ of the nose, auricles, and the mucous membranes of the mouth and tongue are frequent locations, in the latter appearing as excoriated patches from rupture of the vesicles.

Herpes progenitalis; in the male the chief site is the prepuce (*herpes præputialis*). In the female they are comparatively rare; but when occurring, they appear upon the labiæ majora and minora and the skin about the vulva. This variety is preceded by burning, itching, or neuralgic pains and accompanied by redness, congestion, and more or less edema. The importance of this condition resides in the fact that not only may it be readily mistaken for some venereal disease but it may also afford a site for inoculation for some more serious affection.

Treatment.—Ordinary herpes of the face seldom requires treatment, the lesions drying and falling off usually within a week or ten days. The application of dusting powders, cold cream, or boric acid solution often aids in lessening the itching and preventing infection.

In herpes progenitalis, cleanliness is of first importance. The lesions should be carefully washed with boric acid solution and then dusted with calomel, aristol, or similar powders. The parts may be rendered less sensitive in frequently recurring cases by astringent lotions containing tannic acid or zinc sulphate. Circumcision will be necessary when an unusually long prepuce is the cause of the condition. In recurring cases of herpes of the vulva, arsenic, internally, is of benefit.

Herpes gestationis; a rare affection of the skin occurring during pregnancy, consisting of erythema, papules, vesicles, and bullæ, attended by intense burning and itching. It may appear at any time of pregnancy up to the seventh month, and continues until after delivery. It is a variety of dermatitis herpetiformis (*q. v.*).

HERPES ZOSTER.

Synonyms.—Zona; shingles; a girdle; cingulum; intercostal neuralgia.

Definition.—An acute, inflammatory disease, characterized by the development of groups of firm and distended vesicles situated upon inflamed bases corresponding to a definite cutaneous nerve, and accompanied by more or less severe neuralgic pains.

Causes.—The eruption and consequent neuralgic pains are the immediate result of an inflammation of the ganglia or of the nerve-trunks and branches,—*a neuritis,*—probably of the trophic fibers of the affected part; but the cause producing this condition is obscure. Among the many that have been suggested are: cold, injuries to nerves, anemia, malaria, and the medicinal use of arsenic.

Pathology.—An inflammation of either the ganglia, the nerve-trunk, or branches — probably the trophic system — causing the development of vesicles in the lower strata of the rete with the infiltration of serum and inflammatory cells of the papillæ and corium.

Symptoms.—The affection begins with neuralgic pains, either of a burning or lightning-like character, with slight febrile phenomena, followed by the appearance of papulovesicles along the tract of pain; these soon become vesicles situated on bright-red, highly-inflamed bases. The vesicles are about the size of pinheads, or, perhaps, a little larger; usually discrete, although they frequently coalesce, forming irregular patches, appearing in groups until the third to the fifth or even tenth day, when they gradually desiccate, and at the end of the second week nothing remains except occasionally a slight scar, which may disappear or become permanent. When the eruption is at its height, it is perfect in its anatomic formation, each vesicle being well-shaped and seated on a bright-red, inflamed patch of skin, and distended with a translucent, yellowish fluid. The vesicles show no tendency to rupture spontaneously. In rare instances they may become purulent, hemorrhagic, or gangrenous.

The eruption is almost invariably confined to one side (unilateral) of the body, although in rare instances it is seen upon both (bilateral) sides. It is usually found upon well-known nerve tracts. Recurrence is rare. According to the region affected it is termed *zoster capitis, zoster frontalis, zoster faciei, zoster ophthalmicus, zoster auricularis, zoster nuchæ, zoster brachialis, zoster pectoralis, zoster abdominalis, zoster femoralis.*

Diagnosis.—The characteristics of herpes zoster are the pains preceding and accompanying the eruption, the unilateral distribution, and the grouped, tense vesicles showing no tendency to rupture, situated over the course of a cutaneous nerve.

Prognosis.—Most cases terminate in recovery within ten days or two weeks. Neuralgia may follow the disappearance of the eruption. Herpes zoster ophthalmicus may give rise to destructive ocular lesions.

Treatment.—The pain will require the administration of antipyrin, gr. xv (1 Gm.), every three or four hours, phenacetin, gr. v (0.3 Gm.), every three hours, sodium salicylate, gr. x-xv (0.6-1 Gm.), every three hours, or if very severe, morphin, gr. 1/8 (0.008 Gm.), and atropin, gr. 1-100 (0.00065 Gm.), hypodermically, near the lesion. The following combination is sometimes of value:

℞. Zinci phosphidi,
Ext. nucis vomicæ ...āā gr. x   āā 0.6 Gm.
M.—Ft. pil No. xxx.
S.—One every two to four hours. (Bulkley).

Galvanization of the affected nerves is also beneficial, the positive pole being placed over the point of emergence of the nerve, and the negative brushed over the terminal filaments in the skin.

Locally, aristol, boric acid, zinc oxid, and similar powders dusted over the lesions are of value. Flexible collodion, containing morphin, painted over the vesicles serves to protect them and lessen the pain.

LICHEN PLANUS.

Lichen planus is a chronic inflammatory disease of the skin characterized by small, flat, angular, umbilicated, glazed, reddish papules, accompanied by intense itching. The eruption may appear suddenly or gradually and usually appears on the extremities. The lesions vary in size from a pinhead to a pea and tend to occur in patches which often assume a linear form. The papules are flat, angular, glazed, slightly umbilicated, and of a reddish or violaceous color. As the affection progresses scales form on the lesions. Itching is marked. After the eruption subsides, the sites of the lesions remain pigmented for an indefinite period.

Causes.—Unknown. Disturbances of the nervous system, such as result from prolonged mental strain, overwork, etc., are prominent factors in the etiology. The condition is observed with greatest frequency in middle-aged individuals in whom there is marked disturbance of the general health.

Pathology.—The lesions are induced by some neurotic disturbance as yet not well understood. They are situated in the upper part of the corium usually surrounding the sweat-ducts. The earliest step in their production is probably a neuroparalytic hyperemia, after which there is a circumscribed round cell infiltration of the corium with enlargement of the papillæ and proliferation of the cells of the rete.

Diagnosis.—The distinctive features of lichen planus are its chronicity and the shape, size, and color of the lesions.

Prognosis.—The course is essentially chronic, but proper treatment often causes disappearance of the eruption.

Treatment.—Internally; remedies such as arsenic, iron, quinin, strychnin, and cod-liver oil should be administered. Rest is of value. The diet should be regulated and the personal hygiene should receive attention.

Locally, lotions and ointments containing antipruritics should be employed. Tar, mercury, salicylic acid, menthol, and carbolic acid are useful in this connection.

Lichen ruber acuminatus is characterized by the appearance of discrete millet-sized, acuminate, scaly papules. The trunk is the most common situation. The lesions are scattered and show no tendency to grouping. They are localized to the hair-follicles.

The hair-sheaths are changed to funnel-shaped formations, with the wide end external and the narrow end pointed toward the bulb, and the papillæ and contained blood-vessels are enlarged. Itching is not marked. The affection is very chronic and is attended by constitutional disturbances. If left to itself, the disease ultimately terminates fatally. The treatment is similar to that of lichen planus but is seldom of much benefit.

Lichen Scrofulosorum.—A chronic disease of the skin, characterized by the formation of millet-seed-sized, rounded or flattened, pale red or salmon colored, more or less grouped, scaly papules. They are observed most frequently on the trunk in scrofulous individuals and are unaccompanied by itching. The treatment consists largely in the internal use of cod-liver oil, iron, quinin, and strychnin. The cod-liver oil may also be employed locally.

PRURIGO.

Prurigo is a rare chronic inflammatory disease, occurring first in early childhood and lasting indefinitely, characterized by pinhead to lentil-seed-sized, pale, red papules appearing usually on the extensor surfaces of the extremities and accompanied by intense itching. It is seldom seen in America. The cause is unknown. The affection occurs most often among the poor. The outlook is unfavorable.

Treatment.—Every effort should be made to improve the general health and to this end the diet and hygiene should receive attention. Cod-liver oil, iron, manganese, hypophosphites, etc., should be given. Locally, bathing in plain water or medicated solutions, betanaphthol ointment (2 percent in children, 5 percent in adults), sulphur ointment (ʒ i to the ounce), and tar preparations are of great value.

ACNE.

Synonyms.—Acne vulgaris; acne disseminata; varus; stonepock.

Definition.—An inflammation, usually chronic, of the sebaceous glands, characterized by the development of papules, tubercles, or pustules, or by a combination of such lesions, usually in various stages of formation, occurring for the most part upon the face.

Varieties.—*Acne papulosa; acne pustulosa; acne artificialis.*

Causes.—The exciting cause is not well understood; by many observers it is believed to be a microörganism. As predisposing causes may be mentioned puberty, digestive disturbances, constipation, menstrual irregularities, anemia, chlorosis, circulatory disturbances, sedentary life, general debility, and lack of muscular tone. The presence of dust and oil on the face, uncleanliness, contact with tar, and the internal administration of the bromids and iodids in excess are also etiologic factors. Acne may exist alone or be associated with comedo or seborrhea.

Pathology.—An inflammation of the sebaceous gland structure and surrounding tissues. There first occurs retention of the sebaceous secretion, which is soon followed by hyperemia and exudation about the glands and in the gland-wall (*acne papulosa*), and infiltration of the connective-tissue (*acne tubercula*), followed by suppuration (*acne pustulosa*). If the inflammatory action be severe, destruction of the gland with a resulting cicatrix occurs.

Symptoms.—*Acne papulosa* or *acne punctata.* This variety is the earliest stage of the inflammatory action, and is of short duration, soon followed by the development of pus. It is characterized by the occurrence of pin-head to pea-sized, flat, more or less pointed papules, situated about the sebaceous follicles, light in color, with a minute central black point, the opening of the sebaceous duct (*acne punctata*). Pustules are not infrequently observed scattered among the papules. The lesion is unaccompanied with either local or constitutional symptoms. While the forehead is the most frequent seat for this variety, they sometimes are seen elsewhere.

Acne pustulosa. This is the fully-developed affection. It is seen upon the face, neck, shoulders, and back as pin-head to pea-sized, rounded or acuminated pustules, seated upon an infiltrated, reddish base of superficial or deep inflammatory product (*acne indurata*). Scattered among the pustules may be seen numerous papules. There are no constitutional symptoms, nor is pain present unless the pustule be handled.

Acne cachecticorum is that variety observed on the trunk and extremities of cachectic individuals. The lesions are large and indolent.

Acne atrophica is characterized by the formation of small atrophic scars on the disappearance of the lesions, while in *acne hypertrophica,* the scar-tissue is hypertrophic.

Acne artificialis is rather a clinical variety, the result, usually, of large doses of the bromids or iodids, the lesion being identical with that of acne pustulosa.

Diagnosis.—The characteristics of the disease are the course, location, and lesions situated at the sites of the sebaceous glands.

Prognosis.—The affection is essentially chronic, lasting for a number of years. With persistent treatment recovery is rather common.

Treatment.—Before prescribing for any case of the disease, it should be carefully studied as to its etiology, since this bears directly upon the treatment. In most instances, the digestive tract is the subject of various disturbances and internal medication is consequently indicated. The character of the food should be regulated, being careful to eliminate all substances known to be difficult of digestion in the particular case under observation, and especially pastries, gravies, cheese, fried foods, pork, etc., from the diet. Alcoholic beverages should be interdicted and tea and coffee allowed in very moderate quantities. Constipation is common and requires for its relief moderate exercise, abdominal massage, and laxatives in addition to regulation of the diet. Among the laxatives of value in this connection may be mentioned the compound rhubarb pill, the aloin, strychnin, and belladonna pill, calomel, blue mass, cascara sagrada, and the salines. The saline waters such as Hunyadi Janos, Saratoga, and Friedrichshalle are very beneficial. The administration of sodium hypophosphite, gr. x (0.6 Gm.), in solution three times daily after meals is also of value.

Stelwagon employs the following combination:

℞. Sodii benzoat.	ʒ ss-ii	2.-8. Gm.
Tr. nucis vomicæ	fʒ ii	8. Cc.
Ext. cascaræ fld.	fʒ ii-iv	8.-16 Cc.
Tr. cardamom. comp.	q. s. ad f℥ iii	90. Cc.

M. S.—Teaspoonful three times daily in water after meals.

The following mixture known as "mistura ferri acida" is extensively used with success in cases complicated with constipation and anemia:

℞.	Magnesii sulphat. ....℥ i	32.	Gm.
	Ferri sulphat. ....gr. iv-viii	0.25-0.5	Gm.
	Acid sulphuric. dilut. ..fʒ i-ii	4. -8	Cc.
	Aq. menth. pip.. q. s. ad f℥ iv	120.	Cc.

M. S.—Tablespoonful in water half an hour before breakfast.

In cases in which chlorosis or anemia exist, tonics such as iron, arsenic, and manganese should be employed. The citrate of iron and quinin in combination with glycerin is an excellent preparation for these cases. In cachetic or scrofulous individuals cod-liver oil, syrup of the iodid of iron, syrup of the hypophosphites, and similar remedies should be administered. The bichlorid of mercury, gr. 1-100 - 1-60 (0.00065-0.0011 Gm.), three times daily, is of great value as a tonic in many cases. In pustular cases, calcium sulphid, gr. 1-10-½ (0.0065-0.032 Gm.), three times daily, is reputed to be of benefit. Change of occupation frequently aids the treatment materially. Uterine disorders should always receive attention, as they often influence the condition considerably. Other genital conditions acting reflexly should not be neglected. In young adult males, I have seen wonderful improvement follow the passage of a fair-sized bougie once or twice weekly.

Local Treatment.—The objects of the local treatment are to stimulate the sebaceous glands to healthy activity and to remove existing lesions. A form of treatment that has been followed by success in many cases consists in first washing the face every night with very hot water; after the face has partly dried precipitated sulphur is dusted on with a powder puff-ball, and removed in the morning by means of hot water and the face lightly mopped dry.

Hyde recommends evacuating the contents of the lesions by means of a needle, rather encouraging slight bleeding, after which the parts are to be bathed with hot water and while the parts are still wet thoroughly scrubbed with green soap, cleansed with water, dried, and anointed with sulphur ointment. This treatment is very stimulating and is applicable only to deep-seated, indolent lesions. Sometimes the affected areas are decidedly irritated when first seen or become so from treatment. Under such circumstances, a saturated solution of boric acid in alcohol or water, or calamin lotion, should be used.

Usually, when the lesions are seated very superficially the following "lotio alba" will be of benefit:

℞.	Zinc. sulphat,		
	Potassium sulphuret. . āā	gr. xxx	2. Gm.
	Aquæ	f℥ iv	120. Cc.

M. S.—Apply locally at night, washing off the sediment with water in the morning.

In the preparation of this solution, each ingredient should be dissolved separately and then mixed. When completed there should be a white precipitate. The addition of glycerin (♏x to the ounce) will cause the sediment to be held in suspension and often aids in the efficiency of the application. Other preparations of sulphur such as sulphur ointment and paste (ʒ i to the ounce), and Kummerfeld's solution are also of value.

℞.	Sulpt. precip.	ʒ iv	15.5 Gm.
	Pulv. camphoræ	gr. x	0.6 Gm.
	Pulv. tragacanth.	gr. xx	1.2 Gm.
	Aquæ rosæ,		
	Liq. calcis........... āā	f℥ ii	āā 60.0 Cc.

M. S.—Kummerfeld's solution. Apply locally night and morning.

Duhring recommends the use of the following, after washing the parts with hot water:

℞.	Sulphuris præcip.	ʒ j	4. Gm.
	Glycerini	fʒ ss	2. Cc.
	Adeps benzoat.	℥ j	32. Gm.
	Ol. rosæ	♏ iij	0.2 Cc.

M. Ft. unguentum.

S.—To be thoroughly rubbed into the skin at night.

The following is employed extensively in sluggish cases:

℞.	Sulph. precip.	ʒ i	4. Gm.
	Ætheris	fʒ iv	16. Cc.
	Alcohol. ......q. s. ad	f℥ iv	120. Cc.

M. S.—Apply locally twice daily.

Resorcin is often used in the form of an ointment (ʒ i to the ounce). Among other local remedies may be mentioned ichthyol, mercurial preparations, betanaphthol, and salicylic acid. Incision, expression, faradism, massage, and the x-ray are also beneficial in selected cases.

Bartholow used the following method in cases of indurated acne, with success: The sebaceous matter was first dissolved out with—

℞. Liquor potassæ ....fʒ j 4. Cc.
Aquæ destil. ....f℥ j 30. Cc.
M. S.—Apply only to the acne lesions, after which the following ointment should be used:

℞. Plumbi nitrat. ....gr. xv 1. Gm.
Petrolat. ....℥ j 32. Gm.
M. S.—Apply locally twice daily.

ACNE ROSACEA.

Synonyms.—Gutta rosea; rosacea.

Definition.—A chronic hyperemia or inflammatory affection of the nose and cheeks, characterized by redness, hypertrophy of the skin, and dilatation and enlargement of the blood-vessels supplying the part, with the development of more or less acne.

Causes.—The etiology is often obscure. Gastrointestinal disorders, anemia, exposure to heat and cold, uterine disease, puberty, menopause, general debility, seborrhea, nasal disease, and the excessive indulgence in tea, coffee, and alcohol are the most common causes. Continual ingestion of proprietary medicines often induce it. Both sexes may be attacked. The affection usually occurs in middle life.

Pathology.—There first occurs blood stasis in the vessels of the part, producing the undue redness. As a result of the stasis, sooner or later the capillaries are dilated and hypertrophied, and following the interrupted circulation, inflammation of the sebaceous gland (acne) occurs, with the development of papules and pustules. This constitutes the typical acne rosacea. The affection may proceed no further, remaining at this point for years, or, rarely, the pathology of this stage is exaggerated, the involved tissues all hypertrophying, and the connective-tissue undergoing a true hyperplasia, causing increased size and abnormal shape of the nose.

Symptoms.—The onset of the affection is slow and insidious, characterized at first by more or less diffused redness of the part, the color being aggravated by contact with water or cold air. If the nose be the part attacked, it is usually greasy (seborrhœic), and is apt to be cool or even cold. This condition may remain for years, but sooner or later the evidence of dilatation and hypertrophy of the capillaries is apparent by the more decided and permanent redness, and upon close examination the enlarged cutaneous blood-vessels are seen as delicate or coarse red lines, running superficially over the skin in an irregular and tortuous course. Shortly afterwards there are developed upon the hyperemic and hypertrophied skin, papules (acne papulosa) and pustules (acne pustulosa), their number never, however, being very great. This constitutes true acne rosacea. The disease may remain in this state, or, rarely, the cutaneous tissues become greatly hypertrophied, the blood-vessels enormously dilated, the glands enlarged, and the connective-tissue undergoes hyperplasia, resulting in permanent, dark-red, bulky formations, the shape of the nose being contorted into various irregular forms (*rhinophyma*).

Diagnosis.—The affection may usually be distinguished from other affections by the dilatation of the blood-vessels, the acne papules, pustules, and tubercles, and the tendency to overgrowth of the connective-tissue. The course is chronic and ulceration never occurs.

Prognosis.—In the early stages, considerable benefit may be afforded by appropriate treatment. Persistence of the milder forms of the disease can usually be traced to a disinclination on the part of the patient to carry out the treatment. In the occurrence of connective-tissue hypertrophy, the prognosis as to cure becomes less favorable.

Treatment.—As in simple acne, a great portion of the treatment should be directed towards the digestive tract. Tea, coffee, and alcohol should be positively prohibited. The remedies advised in acne vulgaris are also applicable in this condition. Extract of thyroid gland, gr. i-ii (0.065-0.13 Gm.), three times daily, over a long period has been of benefit.

Locally, sulphur preparations, particularly, lotio alba and Kummerfeld's lotion, are especially valuable. Resorcin lotion, gr. v-x (0.35-0.6 Gm.), to the ounce, is of benefit in some cases. The following may be used:

℞.	Hydrargyri chlor. corrosiv. gr. ij	0.13 Gm.
	Petrolat. ....℥ j	32. Gm.

M. S.—Apply thoroughly.

Or, the following, suggested by G. H. Fox:

℞.	Chrysarobini ....ʒ ss	2. Gm.
	Collodii ....f℥ j	30. Cc.

M. S.—Put a brush through the cork and paint lesions every evening.

For the second stage stronger applications are usually required. The dilated capillaries should be incised with a sharp knife, in the hope that adhesive inflammation may close the caliber of the vessels, cold water compresses being used to control the bleeding, a few of the dilated vessels being thus treated every day or two, until all have been incised. Another plan is to paint the affected parts, once or twice a week, with a ten-to-twenty-grain solution of potassium, following its application with an emollient poultice. Electrolysis has also been recommended.

Vleminckx's solution may be employed in some cases:

℞.	Calcis ....℥ ss	16. Gm.
	Sulph. precip. ....℥ i	32. Gm.
	Aquæ ....f℥ x	310. Cc.

M.—Boil to six ounces and filter.

S.—Add one part of the solution to ten parts of water and apply locally.

SYCOSIS VULGARIS.

Definition.—A chronic inflammatory disease of the bearded region, due to invasion of the hair-follicles by pus-producing micro-organisms, characterized by papules, pustules, and tubercles.

Causes.—The affection usually occurs on the upper lip, and is often secondary to nasal discharge. The exciting cause is some form of staphylococcus.

Pathology.—The disease consists of an inflammation within and around the follicles as is shown by the presence of hair in each of the lesions.

Symptoms.—The manifestations consist of pea-sized papules and pustules, each perforated by a hair. The interfollicular spaces are free from involvement by these lesions but may be swollen and infiltrated. The lesions dry, forming crusts and are attended by itching, burning, and slight pain. The hairs remain firmly attached except in the occurrence of marked suppuration. The upper lip is the most common seat but other portions of the beard may be attacked. The affection is, very chronic.

Diagnosis.—Care should be taken not to confuse sycosis vulgaris with tinea sycosis or ringworm of the beard. In the former, there are discrete papules and pustules at the sites of the follicles; the hairs are firmly attached as a rule; the course is chronic; the upper lip is most often involved; and there is no fungus demonstrable.

Pustular eczema may be distinguished from sycosis by its more general distribution (not limited to the follicles), its oozing character, and its diffuse inflammatory base.

Prognosis.—The disease is very chronic and recurrences are common. Persistence in the treatment is usually attended by great benefit.

Treatment.—Local applications are of greatest value. The hairs should be kept very short either by clipping or shaving. When there is marked suppuration the affected hairs should be extracted by forceps. If the surface is acutely inflammatory a saturated solution of boric acid, zinc oxid ointment, or other sedative preparation should be applied. Usually, sulphur ointment (ʒi to the ounce) is of most value. It should be rubbed in freely night and morning. Ammoniated mercury, ichthyol, and bichlorid of mercury are also useful at times.

Lupoid sycosis is a rare form of the disease which terminates in scarring and atrophy of the hair-follicles. It is essentially chronic and seldom responds to treatment.

PSORIASIS.

Synonyms.—Lepra; alphos; psora; English leprosy; dry tetter.

Definition.—A chronic affection of the skin, characterized by reddish, more or less thickened and elevated, dry, inflammatory, and somewhat wrinkled patches, variable as to size, shape, and number, and covered with abundant whitish or grayish colored, imbricated scales.

Cause.—Not known. The source of the affection is, probably limited to the skin itself. Gout, rheumatism, heredity, and parasitic infection are believed to be causal factors. It occurs in the robust and in the feeble, and in both males and females. It usually first appears in early life, and recurs at intervals for years. It is not contagious.

Pathology.—"The disease is essentially a hyperplasia of the normal constituents of the Malpighian layer (mucous layer). The increase takes place chiefly in the interpapillary portion of the layer, the growth of which downward causes an apparent increase in the size of the papillæ of the corium, which, however, on closer examination, are found not to be enlarged. In the later stages of the disease the more superficial blood-vessels of the corium become dilated, a more or less considerable emigration of the white blood-corpuscles takes place, and the immediate neighborhood of the vessels, together with the connective-tissue of the corium, becomes the seat of a round-cell infiltration, which, with the effusion of serum, separates the connective-tissue bundles and fibers into an open meshwork. During the period of disappearance of the disease there is a gradual return to the normal condition, until the hyperplasia, dilatation of the blood-vessels, and cell infiltration has completely disappeared. The hair in psoriasis is affected from the beginning of the disease, hyperplasia of the external root-sheath, the structure corresponding to the Malpighian layer of the epidermis, taking place, with extension of the hyperplastic structure, into the surrounding cutis. The sebaceous and sweat glands are not at any time affected" (Robinson).

Symptoms.—Psoriasis begins as small, reddish spots, of the size of a pin's head, which immediately become covered with scanty or abundant whitish or grayish imbricated scales. The spots gradually increase in diameter by peripheral extension, forming patches of various sizes and shapes.

If an attempt be made to detach one of the scales by means of the finger-nail, it will be found to adhere quite firmly to the skin, and to be about the thickness of a card-board. If the reddish patch thus made bare be pinched up between the finger and thumb, and compared with a similar pinch of the healthy skin, its inflammatory thickening will be discerned. A punctate hemorrhage often follows removal of the scales by scratching. *There is no watery* *discharge at any time.* The skin between the patches is perfectly healthy.

While the anatomic lesions are always identical, the eruption assumes such features, in the size and shape of the patches, to give rise to special names:

Psoriasis punctata. The eruption occurs as small, rounded patches, about the size of a pin's head. This is a rare variety. The lesion rapidly increases in size.

Psoriasis guttata. The eruption occurs in the form and size of drops, and when covered with scales gives the skin the appearance of having been splashed with mortar. A quite frequent variety.

Psoriasis nummularis. The eruption resembles variously sized coins.

Psoriasis circinata. The eruption is about the size of the former variety, the center clearing away, leaving the skin normal, although it may continue to enlarge at the periphery, after the manner of *tinea circinata.*

Psoriasis gyrata. The eruption in wavy lines, of the width of about half an inch, resembling circles and semicircles. This variety is a continuation of the former, from the joining of the patches of psoriasis circinata.

Psoriasis diffusa. The patches of eruption are large and of irregular shape, covering a considerable amount of surface. This variety occurs more frequently on the front of the leg and the outer aspect of the forearm.

Psoriasis palmaris et plantaris. In these lesions the eruption is characterized by larger, thicker, and less lustrous scales, and by the occurrence of deep and painful fissures, from which exudes either a serous or sanguineous fluid.

Psoriasis unguium. In psoriasis of the nails they become thickened, opaque, grayish in color, deeply grooved transversely, and often pitted, and in rare cases the nails are replaced by a scaly incrustation.

Any portion of the body is liable to be attacked with psoriasis, but the elbows, knees, and scalp are involved with greatest frequency. The only discomfort the patient suffers is from the itching, which at times is very severe and distressing. The disease is essentially chronic. Few cases become permanently cured but the affection shows spontaneous improvement in the summer months in many cases. The eruption may partially or completely disappear with or without treatment but recurrence is to be expected.

Diagnosis.—A typical attack of psoriasis presents no difficulty in diagnosis. There are a few affections, however, which may be confounding in irregular cases.

Squamous eczema occuring in patches may be confused with psoriasis. In the former the tendency is to involve flexor surfaces, itching is severe, the patches are irregular and do not clear in the center, there is usually a history of moisture, there are no silvery imbricated scales, and there is decided infiltration and thickening.

Papulosquamous syphilis may be distinguished from psoriasis by its history, concomitant signs, distribution, absence of itching, multiformity of the lesions, scanty scaling, and deep-seated infiltration.

Tinea circinata is characterized by more inflammatory lesions and the presence of the fungus in the scales which are not abundant.

Seborrhea of the scalp and psoriasis of the same region are frequently confounded. In the former the scalp is paler, the scales are finer, smaller, more generally diffused, of a grayish or yellowish color, and greasy, sebaceous character. Psoriasis of the scalp occurs in patches, which are reddish and infiltrated, and there are almost always patches of the disease on other parts of the body.

Prognosis.—Removal of the eruption is by no means difficult. Relapses are common. A permanent cure can never be assured.

Treatment.—The *constitutional treatment* includes attention to the diet and hygiene and the relief of any rheumatic, gouty, or gastrointestinal disorders. The most valuable remedy is arsenic, either in solution or pill form, but it should not be administered when the eruption is markedly inflammatory. Potassium iodid, salicylates, thyroid extract, and the alkalies are of benefit in certain cases.

Locally, the scales should first be removed by bathing or by means of unctuous substances. In the early stage when the symptoms are highly inflammatory, soothing applications are to be employed. Usually stimulation is required and for this purpose tar is of great value. The following is frequently employed:

℞. Olei cadini ....ʒ i 4. Gm.
Petrolat. ....℥ i 32. Gm.
M. S.—Apply locally twice daily.

Or—

℞. Olei cadini,
Olei amygdalæ dulc. .āā ℥ ss 16 Cc.
M. S.—Apply locally twice daily.

Or—

℞. Ung. picis (U. S. P.)...ʒ i 4. Gm.
Petrolat. ....℥ i 32. Gm.
M. S.—Apply locally twice daily.

The following formula suggested by G. H. Fox is of benefit:

℞. Chrysarobini ....gr.x-xx-ʒj 0.65-1.3-4 Gm.
Ætheris et alcoholis..aa q. s. q. s.
Collodii ....f℥ j 30. Cc.
M. S.—Rub the chrysarobin with a little alcohol and ether, and add to the collodion. Apply to the affected patch by means of a camel's hair brush, after removal of the scales.

The objection to chrysarobin in ointment form is that it stains the clothing; the following, however, may be employed with good results:

℞. Chrysarobini ....gr. x-xv-xxx 0.6-1-2 Gm.
Petrolat. ....℥ j 32. Gm.
M. S.—Apply to each spot twice daily.

In using chrysarobin, care should be taken not to have the preparation of too great a strength and not to apply it over too large an area, otherwise a dermatitis may result.

Jamieson recommends the following:

℞. Ammonii carbonat. .....ʒ ij 8. Gm.
Adeps lanæ hydrosus ....ʒ iv 16. Gm.
Cerati galeni ....ad ℥ j ad 32. Gm.
M. S.—Apply three times a day, followed by a warm bath at night.

Among other local remedies of value may be mentioned sulphur, ammoniated mercury, salicylic acid, green soap, pyrogallic acid, and resorcin. The application of the X-ray is of benefit in removing the lesions but has no effect in preventing recurrence.

PITYRIASIS ROSEA.

Synonyms.—Pityriasis maculata et circinata; herpes tonsurans maculosus.

Definition.—An acute, self-limited, inflammatory disease, characterized by the appearance of pinkish or rose-colored macules and maculopapules occurring in oval patches occupying chiefly the trunk and thighs. Many of the patches tend to clear up in the center and spread on the periphery. The central portion of each patch presents a somewhat yellowish appearance while the border is pinkish, elevated, and covered with small scales. Slight itching may be present. The affection may be attended by mild constitutional reaction. The course is from four to eight weeks, the eruption undergoing involution spontaneously and being uninfluenced by treatment. The course, location, and character of the lesions will distinguish this eruption from other circinate diseases such as psoriasis, seborrhoic eczema, syphilis, and ringworm.

DERMATITIS.

Inflammation of the skin as the result of local irritation. The symptoms are the ordinary phenomena of inflammation in general, redness, heat, swelling, pain, and tenderness.

Dermatitis traumatica is that form of the affection due directly to local injury.

Dermatitis calorica is the variety which is produced by exposure to extremes of heat (burns) or cold (frost-bite). Various grades of reaction are observed according to the severity of the exposure. Three stages occur in both forms, erythema, vesication, and gangrene.

Treatment.—For burns, a solution of sodium bicarbonate or carron oil (equal parts of linseed oil and lime-water) is of great value. For frost-bite, first rubbing the part with snow and later applying ichthyol ointment (ʒi to the ounce) is a very efficient mode of treatment.

Dermatitis venenata is the form of the condition that arises from contact with poisonous plants and chemical irritants. The most common variety of this is that following exposure (in susceptible individuals) to the poison ivy (rhus toxicodendron), poison oak (rhus venenata), and poison sumach (rhus diversibola). Among other plants capable of inducing this condition may be mentioned the trumpet vine (teconia radicum), dogwood (cormes florida), common radishes, common field daisy (chrysanthemum leucanthemum), star cucumber (sicyos angulatus), and certain fungi. Among irritant drugs frequently inducing this inflammation are mustard, croton oil, cantharides, iodoform, dye-stuffs, tobacco, arnica, liniments of various kinds, turpentine, acid, alkalies, etc. Any substance employed for the treatment of disease of the skin when used in excess may cause the condition. Individuals engaged in trades necessitating the constant handling of flour, sugar, pastes, and similar substances frequently develop this affection. Excessive exposure to the X-ray or Finsen lamp gives rise to dermatitis. Many other substances, not mentioned here, may induce an artificial inflammation if the integument is susceptible to their influence.

Symptoms.—The earliest manifestation is diffuse erythema. If the irritation was not very severe the affection may subside after the occurrence of erythema and slight swelling. Usually, however, the swelling becomes more intense and innumerable vesicles and blebs form on the affcted regions, accompanied by almost intolerable burning and itching. In the ordinary case due to rhus poisoning, the hands, face, and genitalia are the regions most often involved. These symptoms usually subside spontaneously within a week or ten days, but may be prolonged by continued exposure and improper treatment.

Treatment.—In all cases, the irritant should be withdrawn at once. Sedative lotions are most efficacious in subduing the inflammation. One of the most soothing is a saturated aqueous solution of boric acid containing ten minims (0.6 Cc). of glycerin to the ounce. Sodium hyposulphite solution (ʒ i to the ounce), dilute fluid extract of grindelia robusta (ʒ i to four ounces of water), and lotio nigra and lime-water are also very beneficial. The itching may be relieved by the addition of carbolic acid (10 minims to the ounce) and glycerin (10 minims) to any of the preparations. The great danger lies in overtreating, in an effort to allay the itching and burning.

Dermatitis medicamentosa is the term applied to the various cutaneous manifestations that are due directly to the internal administration of certain drugs. This form is influenced by individual susceptibility, elimination through the skin, large dosage, and long-continued administration.

The bromids produce an eruption consisting of papules and pustules resembling acne in many respects but having a more inflammatory appearance. Occasionally, the eruption consists of macules, bullæ, and even fungating nodules, the latter being most common in children.

The iodids usually give rise to an acneiform eruption but may produce bullous, papular, or erythematous cutaneous manifestations.

Cubebs and copaiba in susceptible individuals give rise to erythema, macules, and papules, the eruption often resembling urticaria or erythema multiforme.

Antipyrin and other coal-tar products are not infrequently followed by morbilliform, erythematopapular, or urticarial eruptions which are prone to itch and desquamate.

Belladonna and its alkaloid atropin occasionally induce a diffuse erythematous eruption on the face, neck, and chest resembling scarlet fever. Associated with it are dryness of the throat, dilatation of the pupils and mild delirium.

Arsenical preparations may give rise to urticarial, erythematous, papular, or vesicular manifestations. Long-continued administration is often followed by pigmentation of the skin.

Chloral occasionally produces an erythematous or an urticarial eruption.

Quinin in susceptible persons may be followed by erythematous, urticarial, purpuric, or vesicular eruptions.

Opium and its derivatives may give rise to pruritus, erythema, papules, or wheals.

Serums employed for antitoxin purposes are not uncommonly followed by urticarial eruptions.

Dermatitis factitia is the term applied to eruptions produced by the patient for the purpose of exciting sympathy or attention. They resemble none of the well-recognized diseases and the diagnosis is always difficult. The lesions usually occur suddenly on accessible regions. The patients are mostly hysteric women or malingerers.

Dermatitis exfoliativa is a very unusual affection, in which the inflammation is attended with high fever and followed by extensive desquamation.

FURUNCULUS.

Synonyms.—Furunculosis; furuncle; boil.

Definition.—An acute affection of the skin, characterized by the occurrence of one or more circumscribed cutaneous or subcutaneous abscesses (boils), which usually terminate by necrosis of the central tissue, with its subsequent expulsion in the form of pus or a core, and a resulting cicatrix.

Causes.—The exciting cause is infection of the hair follicles with pus-producing microörganisms. As contributory causes may be mentioned general debility, anemia, diabetes, uremia, local friction or injury, uncleanliness, and contact with certain irritants, particularly tar and petroleum.

Pathology.—The process resulting in a "boil" has its origin in either a sebaceous gland, a sweat gland, or a piliary follicle, and never begins in the meshes of the corium. "It begins as a small, roundish spot which increases in size until certain dimensions are attained, when it undergoes suppurative change, resulting in the formation of a central point or core, composed of the tissue of the gland in which the furuncle originated, which, together with the pus, is cast off. It shows no disposition to become diffuse, being always a circumscribed inflammation. After the discharge of the core a cavity of more or less depth remains, showing the tissues around it to be hard and infiltrated. After a few days or a week it fills up by granulation, leaving a cicatrix which is often permanent. The central point or core, when thrown off, is composed of a whitish, tough, pultaceous mass of dead tissue, varying in size with the extent and depth of the inflammation" (Duhring).

Hydro-adenitis, as seen in the axillæ, around the nipples, and about the anus or perineum, differs from the ordinary "boil" merely in being deeper seated.

The staphylococcus pyogenes aureus is the microörganism responsible for the condition in most cases. The tricophyton fungus is occasionally the exciting cause.

Symptoms.—"Boils" may occur singly, or more commonly in crops of two, three, or more, another crop following their disappearance (*furunculosis*).

The abscess begins as a small, rounded, imperfectly defined, isolated, reddish spot, of a highly inflamed character, painful on pressure, its size gradually increasing, its central point presenting evidences of suppuration. It reaches its full development in about a week, when it consists of a slightly raised, rounded, and pointed inflammatory swelling, with a yellowish point in the center—the "core." Abscesses with no central suppuration or core are called "blind boils." The size of a developed boil varies from a split-pea to a walnut, the color deep red, with a yellow center, surrounded by a slight areola. The pain of a boil is dull and throbbing, increased on pressure, and usually worse at night. The constitutional symptoms are mild or severe, according to the number and size of the lesions.

Any portion of the body may be attacked; its preference, however, is for the face, neck, back, axillæ, nipples, buttocks, anus, perineum, and labiæ.

Prognosis.—Single lesions usually pass through their course without affecting the general condition of the patient. Furunculosis may be very difficult to relieve and may impair the general health.

Treatment.—In all cases of continuous furuncle formation, the urine should be examined and any organic disease promptly treated. In these cases tonics such as iron, quinin, and strychnin should be administered. It is a common practice, even when the lesions are single, to administer calcium sulphid, gr. 1-10 - 1/8 (0.0065-0.008 Gm.), every two or three hours. The efficicacy of this treatment is doubtful.

Locally, warm applications often aid in hastening suppuration, which when it has occurred indicates early incision to allow expulsion of "the core." Shaving of the hair in the immediate vicinity frequently prevents infection of adjacent follicles. If the lesion is exposed to friction it should be protected by soap-plaster or adhesive plaster. Among the various methods recommended for aborting furuncles may be mentioned crucial incisions, injection of two to five drops of carbolic acid (5 percent solution) into the apex of the boil, and the application of equal parts of glycerin and extract of belladonna or the ointment of nitrate of mercury. Ichthyol ointment (25 percent) is of great value.

CARBUNCULUS.

Synonyms.—Carbuncle; anthrax benigna.

Definition.—An indurated, more or less circumscribed, dark-red, painful, deep-seated inflammation of the skin and subcutaneous connective-tissue, terminating in a slough and the subsequent production of a permanent cicatrix.

Causes.—The exciting cause is some pathogenic microörganism. The affection usually occurs in middle-aged individuals and in men more often than in women. Impairment of the general health, diabetes, and local injury are also factors in its production.

Pathology.—Although Billroth regards furuncle and carbuncle as differing only in degree, the explanation of Warren, of Boston, seems the more probable, he being the first to call the attention of histologists "to the existence of small columns of adipose tissue leading from the panniculus adiposus up to the roots of the lanugo hairs, taking an oblique direction in a line with the erectores pilorum. The inflammation resulting in suppuration of the subcutaneous adipose tissue must either form an abscess or become diffuse. In phlegmonous erysipelas the latter condition is observed; but when the inflammation is in the dermoid texture, the exudates infiltrate the skin and naturally follow the canals occupied by the 'columnæ adiposæ.' The pressure thus exerted upon the whole dermoid tissue cannot fail to strangulate the circulation, and thus produce gangrene of the tissue, even if the exudate be not poisonous enough to destroy the cells by its presence. It can, by this explanation, be easily understood why this disease is apt to affect the skin on the nape of the neck and the back more than on other parts of the body. At this point the skin is dense, its fibrous element extending deep into the adipose layer, which is surrounded by strong bands; hence, the pus confined in such a place, seeking the easiest outlet, will travel along these miniature adipose canals, producing the peculiar appearance pathognomonic of carbuncle."

Symptoms.—The affection is usually manifested by a single lesion which occurs with greatest frequency on the back of the neck, shoulders, or between the scapulæ. It begins in the lower layers of the integument and at first resembles a phlegmon but is devoid of its bright redness. It is rounded in the early stages and vesicles may be present. Soon the affected area becomes firm, circular, flat, and raised above the surrounding parts with painful infiltration of the skin and subcutaneous connective-tissue. The size varies from a hazel nut to an orange and the color is violaceous. After a week or ten days, the constant pressure results in sloughing of the overlying skin at numerous points, through which necrotic masses and purulent material are discharged. This gives the lesion a cribriform appearance which is especially characteristic. Later the entire mass terminates in a slough, which, on being detached, leaves a large, open, deep ulcer, with firm, everted edges, granulating slowly, a permanent cicatrix marking the site of the lesion. The development of the carbuncle is attended by severe pain, of a deep, throbbing, and burning character.

Anorexia, coated tongue, general malaise, and moderate febrile reaction accompany all cases but vary according to the size, number, and severity of the lesions. In very severe cases, symptoms of septicemia are superadded.

Diagnosis.—The characteristics of carbuncle are the single lesion, the size, the phlegmonous nature of the inflammation, the cribriform appearance, the gangrenous termination, and the marked constitutional disturbances.

Prognosis.—The outlook is never very favorable, as general septic infection is liable to occur at all times. It is most serious when occurring in the aged, alcoholics, diabetics, and greatly debilitated subjects and when situated on the upper lip (an unusual location). In ordinary cases, the prognosis is not so grave but the possibility of general infection should always be borne in mind.

Treatment.—Constitutional treatment is of great importance. Nutritious diet, stimulants, and full doses of remedies such as tincture of the chlorid of iron, quinin sulphate, arsenic, strychnin, and ammonium chlorid should be prescribed. Calcium sulphid, gr. 1/8 (0.008 Gm.), every two hours, is of benefit in some cases. Opium, chloral, or phenacetin may be necessary to relieve the pain.

Locally, the injection of a saturated solution of pure carbolic acid through the several apertures of the lesion in every direction through the sloughing tissue, is often very beneficial. It produces severe pain for a short time afterward. The injection of ten to twenty minims of a 5 percent solution of carbolic acid in glycerin, into the lesion very early in its course, may serve to abort it. The application of stick caustic potash directly into the openings of the carbuncle is also of value. Crucial incision may be employed at times. Strapping of the lesion by means of adhesive strips applied in concentric squares, painting with cantharidal collodion or tincture of iodin, or the daily application of nitrate of mercury ointment (U. S. Ph.) may also be used. With the occurrence of necrosis, hot antiseptic solutions should be applied to aid in separation of the gangrenous slough. If septicemia threatens the necrotic tissues should be excised.

PARASITIC DISEASES.

TINEA CIRCINATA.

Synonyms.—Tinea trichophytina corporis; herpes circinatus; ringworm of the body.

Definition.—A contagious, vegetable parasitic affection of the skin, due to the trichophyton fungus, characterized by the development of one or more circular or irregularly shaped, variously sized, inflammatory, slightly vesicular or squamous patches, occurring upon the general surface of the body.

Causes.—Ringworm of the body is caused by the presence of a vegetable parasite discovered by Bazin, in 1854, termed the *trichophyton,* the same growth or fungus that produces tinea tonsurans and tinea sycosis. The affection is highly contagious, and is frequently communicated from one individual to another, although it has been determined that a certain unknown condition of the skin is requisite for its development. In children it is most frequently seen among the weakly and the poorly nourished. In adults it is usually associated with a decline in the general health.

Pathology.—The fungus is seated between the strata of the epidermis, more particularly in the superior layers of the rete. Mycelium, consisting of long, slender, jointed threads may be found in abundance but spores are very scant. The presence of this foreign body produces the subsequent phenomena—a superficial dermatitis, erythema, exudation, minute vesiculation and papulation, and, in the severe grades, tubercles and pustules. The desquamative symptoms are exfoliative—nature's efforts for relief.

Symptoms.—Tinea circinata varies greatly in the degree of its development, from the trivial complaint so often seen in children, to the chronic, extensive, and obstinate disease sometimes seen about the thighs in adults (*tinea circinata cruris*).

The disease usually begins as a small, reddish, scaly, rounded or irregularly shaped spot of papules, which in a very few days assumes a circular form (ringworm). It continues to increase in size, the papules often changing to vesicles. A characteristic of the eruption is its healing in the center as it spreads on the periphery. Occasionally the circles or rings coalesce, forming serpiginous lesions. The usual size of a fully developed ringworm is about that of a silver quarter of a dollar. The affection occurs with greatest frequency upon the face, neck, and backs of the hands. Itching is slight as a rule.

Chronic tinea circinata often lacks the characteristic annular configuration but instead appears in the form of single or multiple, disseminated, small, reddish, slightly scaly, ill-defined spots which may or may not be elevated above the surrounding skin. The size varies, and the line of demarkation between the lesion and the healthy skin may be lacking.

The "eczema marginatum" of Hebra is to be looked upon as a severe form of tinea circinata.

Tinea circinata cruris, or ringworm of the thighs, a variety of the "eczema marginatum" of Hebra, is usually complicated with true eczema, and is a very obstinate, chronic form of the affection; it is accompanied by severe itching.

Tinea trichophytina unguium is a variety. The nails become opaque, whitish, thickened, and soft and brittle, especially along their free border. Its course is chronic, and it is difficult to cure.

Diagnosis.—While in many cases, the history, course, character of the eruption, etc., will serve to distinguish tinea circinata from other circinate eruptions, the diagnosis should always be rendered positive by a microscopic examination of the scales removed from the lesion. The scales should be placed upon a glass slide containing a drop of liquor potassæ over which is laid a thin glass cover. After remaining for a few minutes, the fungus may be detected by a microscope having a magnifying power of from 250 to 500 diameters.

Prognosis.—The affection is usually very amenable to treatment but occasionally it exhibits great obstinacy. At times relapses occur.

Treatment.—Local applications usually suffice to cure the affection. The patch should be washed with soap and water and one of the following supplied:

℞.	Cupri acetat. ....gr. x	0.6 Gm.
	Ung. aquæ rosæ ....℥ j	32. Gm.

M. S.—Apply locally twice daily.

Or—

℞.	Hydrargyri ammoniat. ..gr. xx-xxx	1.3-2. Gm.
	Petrolat. ....℥ j	32. Gm.

M. S.—Apply locally twice daily.

Or—

℞.	Hydrargyri chloridi cor. .gr. j	0.065 Gm.
	Tinct. benzoin. comp. ..f℥ j	30. Cc.

M. S.—Apply locally twice daily.

Or—

℞.	Sulph. precip. ....ʒ i	4. Gm.
	Acid. boric. ....ʒ i	4. Gm.
	Petrolat. ....℥ i	32. Gm.

M. S.—Apply locally twice daily.

Or—

℞.	Sodii hyposulphit. ......ʒ i	4. Gm.
	Aquæ ....f℥ i	30. Cc.

M. S.—Apply locally twice daily.

Among other remedies of value may be mentioned tar, resorcin, betanaphthol, chrysarobin, protargol, and sublamin.

In obstinate cases of tinea cruris, the parts should be treated with a saturated solution of boric acid and afterward covered with boric acid powder or ammoniated mercury ointment (gr. xxx to ℥ i).

TINEA TONSURANS.

Synonyms.—Tinea trichophytina capitis; herpes tonsurans; ringworm of the scalp.

Definition.—A contagious, parasitic affection of the scalp, due to the trichophyton fungus, characterized by the development of circumscribed, vesicular or squamous, more or less bald patches in which the hair is diseased and usually broken off close to the scalp.

Cause.—It results from the presence and growth of the same fungus giving rise to tinea circinata—*trichophyton*. It is an affection of childhood, seldom being seen after puberty. It is highly contagious, and may be contracted from a case of ringworm of the body.

Pathology.—The fungus invades the hair, hair-follicles, and adjacent epidermis causing disintegration of the hair and distention of the follicle which becomes prominently raised. Spores are present in abundance but the mycelium is very scant. The hair-shaft is fractured just above the level of the scalp, and usually presents a jagged, bristly, stubble-like extremity. The epidermis of the scalp may either present minute vesicles and desquamation, or, in severe cases, edema and inflammatory symptoms, with fluid exudation (*tinea kerion*).

Symptoms.—Ringworm of the scalp usually begins in the form of small circumscribed patches, which soon become the seat of small vesicles or pustules, terminating in desquamation, or of furfuraceous scales. The patches spread rapidly, soon reaching the size of a silver quarter to that of a silver dollar. They are circular in form, circumscribed, of a reddish, grayish, or greenish-yellow color and covered with fine or coarse scales, with the hairs broken off close to the scalp. The epidermis of the scalp is more or less raised, and the follicles are prominent, giving the characteristic appearance of the disease—the goose-skin or plucked-fowl appearance. As a result of the loss of hair, baldness, more or less complete, but temporary, exists. Itching is a constant symptom.

Ringworm of the face or body (*tinea circinata*) may complicate tinea tonsurans.

Tinea kerion is a severe variety of tinea tonsurans, "characterized by edema, inflammation, and the exudation of a viscid, glutinous, yellowish secretion from the opening of the hair-follicles. When fully-developed the patches are yellowish, reddish, or purple in color, and are more or less raised, edematous, and boggy. They are uneven and honeycomb-like (hence the name kerion), and studded with yellowish, suppurative points, or, later with small cavities or foramina, the openings of the distended hair-follicles deprived of their hairs, which discharge a mucoid, gummy, honey-like fluid." The patches are tender, painful and at times the seat of itching.

Diagnosis.—The affection is usually readily differentiated from other diseases of the scalp by its occurrence in children, in the shape of circumscribed, sharply marginated, more or less circular patches of incomplete baldness characterized by broken-off hairs, very prominent follicles, and grayish scales. The presence of the fungus is diagnostic. A hair should be extracted, and examined after being immersed in liquor potassæ.

Prognosis.—The disease is essentially chronic. If untreated it may persist for two or three years or until puberty, when its soil seems to be exhausted and the affection subsides. Even under treatment the patches may last for six months or more.

Treatment.—Local treatment only is required, and it should be vigorous and persistent. No case should be discharged until the microscope shows absence of the fungus in the extracted hairs. Mild cases should be treated by cutting the hair as close as possible and thoroughly scrubbing the patches with green soap and water or by the application of a 25 to 50 percent solution of boroglycerin, twice daily, or a 6 percent solution of the oleate of mercury, or one of the following:

℞.	Sulph, precip.	ʒ i	4. Gm.
	Petrolat.	℥ i	32. Gm.

M. S.—Apply locally twice daily.

℞.	Betanaphthol	ʒ i	4. Gm.
	Petrolat.	℥ i	32. Gm.

M. S.—Apply locally twice daily.

℞.	Protargol	gr. xxiv	1.5 Gm.
	Petrolat.	℥ i	32. Gm.

M. S.—Apply locally twice daily.

℞.	Sodii borat.	ʒ i	4. Gm.
	Aquæ destil.	f℥ ii	60 Cc.

M. S.—Apply thoroughly several times daily.

℞.	Acid. boric.	gr. xv	1. Gm.
	Sulph. flor.	gr. xv	1. Gm.
	Petrolat.	℥ iss	47. Gm.

M. S.—Apply to scalp night and morning.

℞.	Cupri oleat.	ʒ ss	2. Gm.
	Petrolat.	℥ ii	63. Gm.

M. S.—Apply locally after washing the scalp with boric acid solution.

℞.	Thymol	ʒ ss	2. Gm.
	Chloroformi	fʒ ij	8. Cc.
	Ol. olivæ	fʒ vj	24. Cc.

M. S.—Morris-thymol solution. Apply locally.

A preparation very popular in London, known as Coster's paste, is used by painting the patches with a brush and allowing it to remain on until the crust is cast off in the course of five or six days, when it may be reapplied. A few applications often suffice. Its formula is—

℞.	Iodin	ʒ ij	8. Gm.
	Olei picis	f℥ j	30. Cc.

M. S.—Apply as directed.

An excellent application in rebellious cases is—

℞.	Potassæ (caustic)	gr. ix	0.6 Gm.
	Acid. carbolici	gr. xxiv	1.5 Gm.
	Adeps lanæ hydrosus	℥ ss	15. Gm.
	Ol. theobromæ	f℥ ss	15. Cc.

M. S.—A small amount should be rubbed into head night and morning. If the scalp is not shaved, the application is retained better.

Another excellent application is—

℞.	Ung. acid. borici	℥ ij	63. Gm.
	Ung. eucalyptol	℥ ij	63. Gm.
	Ol. caryophylli	fʒ ss	2. Cc.
	Glycerini	q. s.	q. s.

M.—Ft. ung.

S.—Apply locally.

Cases which resist these means are to be treated by removing the loose hairs about the edges of the patches and the broken-off hairs over the surface, by means of small, broad-bladed, short forceps, a few hairs only being seized at a time, a portion of the diseased hairs being removed each day until the surface has been cleared. After each depilation one of the above formulæ should be applied.

TINEA SYCOSIS.

Synonyms.—Tinea trichophytina barbæ; sycosis parasitica; barbers' itch; ringworm of the beard.

Definition.—A contagious, parasitic affection of the hair, hair-follicles, and subcutaneous tissues of the hairy portion of the face and neck in the adult male, due to the *trichophyton fungus;* characterized by the development of tubercles and pustules.

Cause.—The direct cause is the tricophyton fungus. Its growth is no doubt aided by some ill-defined, impairment of the integument. The affection is usually acquired in the barber shop but may be contracted from the lower animals, especially horses and cattle, in which cases it is of unusual intensity.

Pathology.—The parasite finds its way into the hair-follicles and attacks the root and shaft of the hair, causing inflammation, followed by more or less follicular suppuration and general infiltration of the surrounding tissues. The presence of the fungus also results in inflammation of the subcutaneous connective-tissue giving rise to the well-known tubercular formations peculiar to the affection. They are firm, comparatively painless, and manifest but little disposition to undergo change, remaining during the presence of the fungus and finally disappearing gradually without leaving a scar. Under the microscope the parasite is plainly discernible.

Symptoms.—Barber's itch begins as an attack of tinea circinata, with one or more reddish, scaly patches. Soon the redness and desquamation become more marked and swelling and induration occur. With the advance of the disease, the hairs become dry, brittle, and loose. The skin soon becomes distinctly nodular and lumpy, and pustules develop about the openings of the follicles. The subcutaneous tissue is also involved, giving rise to thick, firm masses of induration. The affected area has a dark-red or purplish color and is studded with a large number of tubercles and pustules. Many of the pustules discharge a purulent material which accumulates forming crusts. The hairs are always diseased and either drop out or break off in the follicles or just above the level of the surface. The chin, neck, and submaxillary region are the most frequent situations of the disease. Itching, burning, and pain, of varying severity, are always present. The affection is extremely chronic and relapses are common.

Diagnosis.—While the diagnosis can always be made with certainty by the aid of the microscope, the affection presents certain clinical characteristics that are distinctive in very many cases.

In tinea sycosis, or sycosis parasitica, the skin and subcutaneous connective-tissue are extensively involved, as manifested by the induration and formation of the characteristic tubercles. The upper lip is rarely invaded; the hairs are diseased, broken off, or loose, and under the microscope reveal the parasite.

Sycosis non-parasitica is a chronic, inflammatory, *non-contagious* affection of the hair-follicles, characterized by the development of papules and pustules which are perforated with hairs, the hairs themselves being unaffected. The upper lip, cheeks, and chin are the parts mostly involved. If of long duration, some inflammatory thickening results.

Pustular eczema may resemble tinea sycosis, with extensive pustulation and crusting; but in the former the hairs are not involved, nor are the characteristic tubercles present.

Treatment.—The following plan of treatment is very effective: Any existing crusts should first be thoroughly saturated with almond or olive oil and removed by washing with soft soap and water. The part is then cleanly shaved, the first operation being more painful than subsequent ones. After shaving, the face is bathed in water as hot as can be borne. All pustules should then be opened with a fine needle and the parts sponged freely with a solution of sodium hyposulphite ʒ i (4 Gm.), water f℥ i (30 Cc.), after which the parts are again thoroughly washed with hot water, carefully dried, and sulphur ointment, ʒ i-ii (4.-8. Gm.) to the ounce (32. Gm.) applied. This procedure should be performed preferably at night. The following morning the ointment is washed off with soap and water, the face bathed with the sodium hyposulphite solution, and dusted with any inert powder. This plan of treatment should be continued regularly every night, omitting the shaving when the beard is not sufficiently long to permit it without great distress. In very obstinate cases depilation should be practiced, alternating with shaving. The various applications recommended in the treatment of tinea tonsurans are also applicable to this form of the disease.

TINEA VERSICOLOR.

Synonyms.—Pityriasis versicolor; liver-spots; chromophytosis.

Definition.—A contagious parasitic affection of the skin, due to the *microsporon furfur,* characterized by the occurrence of variously sized, irregularly shaped, dry, slightly furfuraceous, yellowish spots upon the chest or other portions of the body.

Cause.—Pityriasis versicolor is the result of the presence upon the surface of the skin of a vegetable fungus termed *microsporon furfur.* It is a mildly contagious affection seen after puberty. It is said to occur most frequently in those suffering from wasting diseases, particularly phthisis pulmonalis. It is not connected with any affection of the liver, as supposed by the laity.

Pathology.—The fungus permeates the horny layer of the epidermis, never the hairs or nails, and gives rise to the irregularly shaped and sized macules of a yellowish or brownish color. The fungus consists of short, jointed, angular mycelial threads and rounded spores more or less grouped. As a rule, it gives rise to neither hyperemia nor inflammatory symptoms.

Symptoms.—Tinea versicolor occurs in the form of irregular, roundish, circumscribed, or reticulated macules. The spots vary in size from that of a small silver coin to that of the hand. By coalescing they often cover a greater portion of the chest, their most usual site. Upon close inspection the surface of the macule is seen to be covered with furfuraceous scales, and, if the scales be not visible, scraping with the finger-nail will demonstrate their presence. In color the spots vary from a delicate buff or fawn shade to a yellowish, deep brown, and, rarely, even blackish hue. At times mild itching accompanies the eruption. The affection is chronic and in the absence of treatment, persists indefinitely. Response to treatment, however, is prompt, but relapses are frequent, due in all probability to a failure to continue treatment for a sufficient period.

Diagnosis.—The history, course, location, and character of the eruption are distinctive enough to prevent error but in doubtful cases resort should be made to the microscope.

Treatment.—The parts should be thoroughly cleansed with soap and water and either of the following lotions applied:

℞. Sodii hyposulphitis	ʒ iij	12. Gm.
Glycerini	fʒ ij	8. Gm.
Aquæ	ad ℥ iv	ad 120 Cc.

M. S.—Apply frequently.

Or—

℞. Hydrargyri chlorid. corrosiv.	gr. iv	0.26 Gm.
Alcoholis	fʒ vj	23. Cc.
Ammonii chlorid.	ʒ ss	2. Gm.
Aquæ rosæ	ad f℥ vj	178. Cc.

M. S.—Apply frequently. (Tilbury Fox).

TINEA FAVOSA.

Synonyms.—Favus; porrigo favosa; honeycombed ringworm; crusted ringworm.

Definition.—A contagious affection of the skin, due to a vegetable parasite—*Achorion Schönleinii;* characterized by the development of either discrete or confluent, small, circular, cup-shaped, pale-yellow, friable crusts, usually perforated by hairs.

Cause.—The presence and growth of a vegetable parasite known as the *Achorion Schönleinii* is the cause of tinea favosa. It is more common in children than in adults, attacking the former in the first place either *de novo* or through direct contagion, and is from them communicated to adults. The affection is often contracted from the lower animals. It is a disease confined almost exclusively to the lower classes, especially, of Russians, Polish, Austrians, and Hungarians.

Pathology.—Tinea favosa may have its seat either in the hair-follicles and hair, or upon the surface of the skin or the nails; the former, however, being the structures most frequently involved. It is purely a local affection, due solely to the presence and growth of the vegetable parasite discovered by Schönlein, of Berlin, in 1839, and named after him—*Achorion Schönleinii.* The crusts are made up almost entirely of fungus, which upon section is seen with the naked eye to be composed of a porous mass and to possess a pale-yellow or whitish color. Under the microscope it is seen to consist of both mycelium and spores in great quantity and in all stages of development.

Symptoms.—When the affection attacks the hairs and follicles it is termed *tinea favosa pilaris;* when the epidermis, *tinea favosa epidermis;* and when the nails, *tinea favosa unguium.* Rarely all the structures may be attacked at one and the same time; its usual seat, however, is the scalp. The disease begins by the development of one or of several *pin-head sized, pale-yellow crusts,* seated about the hair-follicles. In about a fortnight these crusts have increased in size and are umbilicated, *favus cups,* circumscribed, circular in form, friable, and very slightly elevated above the level of the skin. Ordinarily, they are of a pale-yellow or sulphur-yellow color, but after a time, from dust and other matters, they become brownish or greenish yellow in color. The number of crusts vary from very few to immense numbers. The usual size is about that of a split pea. In *tinea favosa pilaris et capitis* the affection is often accompanied by pediculi, while swelling of the glands of the neck and small abscesses upon the scalp are not uncommon. The hairs become lusterless, opaque, brittle, and at times split longitudinally, and from atrophy of the follicles and sebaceous glands and scarring, permanent baldness may result.

The lesions have a peculiar odor, resembling that of mice, or of musty, stale straw.

In *tinea favosa unguium* the nails become thickened, yellow, opaque and brittle.

Diagnosis.—The distinctive features of this disease that will serve to differentiate it from other affections of the scalp are its history, long duration, the sulphur-yellow, umbilicated, crusts, the peculiar odor, the atrophic scarring, and the presence of the fungus which may be readily detected by microscopic examination.

Prognosis.—Tinea favosa of the epidermis readily responds to treatment. Tinea favosa pilaris is more obstinate, and if of long duration, may result in baldness.

Treatment.—Many of the patients are in comparatively poor health and require general tonic treatment. Attention to personal hygiene and cleanliness should not be neglected. The local treatment is of great importance and consists essentially in depilation and the application of parasiticides. The hair should be cut off as short as possible, the crusts removed by the use of oil, or soap and hot water, or poultices, again well oiled, and the hairs removed by means of broad-bladed forceps, a few hairs being removed at a time and only a small surface cleared at each sitting, after which the following lotion is to be thoroughly applied:

℞.	Hydrarg. chlorid. corrosiv.gr. v-x	0.3-0.6	Gm.
	Ammonii chlorid. ....ʒ ss	2.	Gm.
	Misturæ amygdalæ amar.f℥ iv	120.	Cc.

M. S.—Apply thoroughly. (Bulkley).

Stelwagon employs the following:

℞.	Acid. carbol. ....ʒ i	4. Gm.
	Ung. picis. liq.,	
	Ung. hydrarg. nitrat. ,āā ʒ ii	8. Gm.
	Ung. sulphur. ....ʒ iv	16. Gm.

M. S.—Apply locally.

Owing to the decomposition likely to occur in this preparation it should be prepared freshly or within a week of its being employed.

Shoemaker recommends the application of oil of ergot for twenty-four hours to soften the crusts, after which boroglycerin (25 to 50 percent) should be sponged thoroughly over the affected surface; in a few hours the crusts will peel off and the surface can be cleansed after which the following should be applied:

℞. Ung. hydrargyri oleat. ..℥ ss 16. Gm.
Adeps ....℥ ss 16. Gm.
M. S.—Apply a small portion to each cup daily for two or three days.

Then alternate with—

℞. Cupri oleat. ....ʒ ss 2. Gm.
Adeps ....℥ j 32. Gm.
M. S.—Small portion to the affected spots.

The application should be made every day or two and continued for a period of three or four weeks. If after an equal interval the disease is still present, the treatment is renewed.

Other parasiticides may also be employed, the keynote to success in their use is regular vigorous application continued over a long period. No case should be discharged until the microscope is no longer able to reveal the fungus.

When the nails are affected they should be scraped in addition to the local applications.

SCABIES.

Synonym.—The itch.

Definition.—A contagious, animal parasitic disease of the skin, due to the *acarus,* or *sarcoptes scabiei;* characterized by the formation of cuniculi (burrows), papules, vesicles, pustules; followed by excoriations, crusts, and general cutaneous inflammation, and accompanied by itching.

Causes.—The essential cause is the animal parasite, *acarus* or *sarcoptes scabiei.* The affection is contagious and attacks individuals at all ages and in every walk of life. It may be contracted by direct contact with infected persons or through the medium of bed-clothes and similar articles. It is most frequent where there are large bodies of people congregated together under unhygienic conditions, as in camps, barracks, ships, tenement houses, etc.

Pathology.—Scabies is an inflammation of the skin with the development of papules, vesicles, pustules, excoriations, and subsequent crusting, the result of the ravages of the animal parasite, together with the irritation produced by the scratching of the patient.

The parasite *acarus*, or *sarcoptes scabiei*, is a minute creature, barely visible to the naked eye, appearing as a yellowish-white, rounded body. The female is the most commonly encountered; the males are said to take no part in causing the affection, and are rarely seen. They are said to die in about a week after copulation with the female. The female finds her way boring through the horny layer into the mucous layer of the epidermis, and, being impregnated, begins at once laying her eggs and at the same time making her burrow. A variable number of eggs are deposited, usually about a dozen, after which she perishes in the skin. The ova hatch out in six or ten days.

Symptoms.—The eruption of scabies is an artificial dermatitis or eczema, according to the amount of irritation produced by the presence of the parasite and the traumatism resulting from the severe scratching of the patient.

Immediately upon the arrival of the itch mite upon the skin it begins its work of burrowing, and very soon a burrow, or *cuniculus*, is formed, in which the eggs are deposited, and which also becomes the habitat of the female during the remainder of her life. The ova are hatched in about one week after their deposit, and at once begin to care for themselves and to burrow, resulting in the formation of as many additional *cuniculi* as there are active female mites. It is the presence of these burrowing parasites that constitutes the irritation resulting in the inflammation of the skin, characterized by the formation of minute papules, vesicles, and pustules, with more or less inflammatory induration. Add to these the excoriations, scratch marks, fissures, torn vesicles, and pustules with yellow and bloody crusts, caused by the scratching, and a picture of the fully-developed disease is seen.

The *burrow*, or *cuniculus*, as it is termed, is formed by the mite entering and making its way beneath the horny layer of the epidermis, which is raised, very much as a mole undermines the ground. It occurs as a slight linear elevation of the epidermis, varying from a half a line to four or five lines in length, and having an irregular or tortuous course. Its color is whitish or yellowish, speckled here and there with dark dots. At either end the cuniculus terminates as darkish points, the more prominent of which represents the parasite.

The papule is the first inflammatory lesion; they are numerous and of small size, and may be the extent of the disease. The vesicles are the next stage, varying in size and number, having an inflamed base, sometimes presenting cuniculi upon their summits. The pustules represent the completion of the inflammatory action, their size and number varying with the severity of the irritation.

The intense itching, which is worse at night, results in excoriations, torn papules, vesicles, and pustules, followed by crustings, which after a time disguise the characteristic lesions. The regions of the body attacked by the parasite are the hands, especially the sides of the fingers and the folds where they join the hands. After a time the wrists, penis, and mammæ, and around about and upon the nipples, are invaded. The resultant multiform eruption is usually found in the various flexor regions of the body, inner sides of the thighs, and the buttocks, but may be general. The face is free from the disease except occasionally in nursing infants. In very clean persons, or those having their hands constantly in water, there may be no burrows or other lesions on the hands.

Diagnosis.—The presence of the itch mite and its burrows is pathognomonic. A multiform eruption most marked in the flexor regions with intense itching, worse at night, and a history of contagion is also diagnostic of the disease. Frequently the burrows are removed by scratching and a careful search fails to detect any of them.

Prognosis.—The disease never tends toward spontaneous cure. When severe, a diffuse eczema may be engrafted on the original condition. Under appropriate treatment response is prompt and cure is rapid.

Treatment.—In every instance, all members of the household having the affection should be treated at the same time. The bed clothing and underclothing of infected individuals should be sterilized. Patients under treatment should sleep alone for obvious reasons. The treatment should be directed first towards the scabies, after which the attendant dermatitis should receive attention. The following plan of treatment is very satisfactory: An ointment such as—

℞.	Sulph. precip. ....ʒ i	4. Gm.
	Petrolat. ....℥ i	32. Gm.

M. S.—Apply locally at night.

should be applied all over the body from the neck to the soles of the feet for four nights, after which a hot bath is taken and the bed-clothes and underclothes are changed. The treatment is then withheld for an equal period to allow the irritation to subside. If after this period elapses the generalized itching returns, the ointment is again applied in a similar manner for three or four days. At the end of this time, only a few localized areas of itching will remain, which will also disappear with the application of a weak carbolic acid lotion or ointment (gr. v to x to the ounce).

Another valuable method of treatment consists in the patient first washing himself thoroughly with soft soap and water, after which a warm bath is taken. Tincture of benzoin or one of the following is then applied all over the body twice daily except the head and face.

℞.	Sulph. precip.,	
	Betanaphthol ....āā ʒ ss	2. Gm.
	Petrolat. ....℥ i	32. Gm.

M. S.—Apply locally.

℞.	Styracis liquid. ....fʒ ii	8. Gm.
	Ung. sulphur. ....ʒ ii-iv	8.-16. Gm.
	Petrolat. ....q. s. ad ℥ i	32. Gm.

M. S.—Apply after washing. (Bulkley).

℞.	Sulph. precip. ....ʒ i	4. Gm.
	Balsam. Peruviani ......ʒ ss	2. Gm.
	Adeps ....℥ i	32. Gm.

M. S.—For children. (Duhring).

℞.	Creolin ....gr. v	.32 Gm.
	Petrolat. ....℥ i	32. Gm.

M. S.—Apply locally.

In children, the strength of all remedies employed for this purpose should be reduced to prevent undue irritation. Styrax and balsam of Peru are most useful in infantile cases. In using beta-naphthol, its stinging properties when first applied should be borne in mind. Apart from this, it is perhaps the most elegant of all these preparations. Care should always be taken not to continue the treatment for too long a period at a time.

PEDICULOSIS.

Synonyms.—Phthiriasis; morbus pedicularis; lousiness.

Definition.—A contagious, animal parasitic disease of the head, body, or pubes, due to the presence of pediculi and characterized by the wounds inflicted by the parasite, together with excoriations and scratch marks.

Varieties.—*Pediculosis capitis; pediculosis corporis; pediculosis pubis.*

Pathology.—The lesion produced by the presence of the pediculi is a minute hemorrhage, caused by the parasite inserting its sucking apparatus, or, as it is termed, its haustellum, into a follicle, and obtaining blood by a process of sucking, and not by biting, as is generally supposed. The presence of the parasite in any great numbers brings about a peculiar irritable state of the skin, which gives rise to an irresistible desire to scratch, as a consequence of which the surface is markedly excoriated.

Symptoms.—The symptoms which arise from the presence of the parasite in different localities are somewhat different, and call for separate consideration.

Pediculosis capitis. This variety is caused by the presence of the pediculus capitis, or head-louse. The *ova,* or *nits,* are readily recognized at a distance. Their favorite seat is the occipital region, either upon the surface of the scalp or upon the hair. Their presence gives rise to considerable irritation, itching, and consequent scratching, resulting in the wounding of the scalp, with oozing of a serous or purulent fluid mixed with blood, which soon mats the hair and forms into crusts. In those predisposed to eczema the presence of the parasite will give rise to that condition.

Pediculosis corporis. This variety of the pediculosis is caused by the presence of the pediculus corporis, or body-louse, or more properly termed the pediculus vestimenti, or clothes-louse. Its color, when devoid of blood, is dirty-white or grayish, with a dark line around the margin of its abdomen. Its habitat is the clothing covering the general surface, remaining upon the skin only long enough to obtain sustenance. The ova are usually deposited in the seams of the clothing, the lice being hatched within the week. Occasionally a few of the pediculi may be observed crawling about the surface, or in the act of drawing blood. As they move over the surface they give rise to an intensely disagreeable itching sensation, to relieve which the patient scratches, which in turn gives rise to the characteristic lesions of the affection.

The lesions are numerous. The scratch marks are scattered here and there, either long and streaked, in other places short and jagged, the excoriations and blood-crusts varying in size from a pin-head to a split pea or even larger, with irregularly-shaped pustules. In addition to the lesions resulting from the scratching are seen the primary lesions, consisting of minute, reddish puncta with slight areolæ, the points at which the parasite has drawn blood. In cases of long standing a brownish pigmentation of the whole skin may result from the long-continued irritation and scratching. The favorite sites of the lesions are the back, especially about the scapular region, the chest, abdomen, hips, and thighs. Pediculosis is seen most commonly among the poorer classes, and especially the middle-aged and elderly.

Pediculosis pubis. This variety of pediculosis is caused by the presence of the pediculus pubis, or crab-louse. Although having its seat of predilection about the pubes, it may also infest the axillæ, sternal region in the male, beard, eyebrows, and even the eyelashes.

They may be found crawling about the hairs, but more commonly hugging the surface closely. They infest adults chiefly, and occasion symptoms similar to those described in connection with the other varieties. They are usually contracted through sexual intercourse, although occasionally they are present in cases in which they have not been communicated in this way, and in which no explanation as to the mode of contagion can be suggested. The itching varies from slight to severe.

Diagnosis.—When violent itching exists in any case, without a well-defined eruption, the possibility of the presence of pediculi should always be entertained, and if carefully sought after, are usually found.

Prognosis.—Favorable, if the treatment be thoroughly carried out.

Treatment.—*Pediculosis capitis.* The most effective application to this variety is to thoroughly soak the head two or three times a day with ordinary petroleum or kerosene oil and keep it wrapped in a cloth for twenty-four hours. At the end of this time the head should be thoroughly washed with soft soap and hot water, dried, and saturated with the official ointment of ammoniated mercury. If required, this entire procedure may be repeated, but usually any pediculi escaping the petroleum are destroyed by the ointment.

Pediculosis corporis. In this variety the habitat of the parasite being the clothing, they must be boiled or baked at a temperature sufficiently high to destroy their life. After this the clothing should be changed every day or two, carefully inspected, and if pediculi are seen, they must again be baked or boiled. For the irritation, itching, and excoriations, mild alkaline baths or lotions of carbolic acid are sufficient.

Pediculosis pubis. The parts should be washed twice daily with soft soap and water, after which the thorough application of tincture of cocculus indicus (fish berries), full strength or diluted, dried, and saturated with the officinal ointment of ammoniated mercury, or mercurial ointment (blue ointment), will be effectual.

HYPERTROPHIES OF THE SKIN.

LENTIGO.

Synonym.—Freckles.

Definition.—A pigmentary deposit of the skin, characterized by irregularly shaped, pin-head or pea-sized, yellowish, brownish, or blackish spots occurring for the most part about the face and back of the hands.

Cause.—In the majority of instances exposure to the sun is the exciting cause.

Pathology.—In anatomic structure freckles consist of a circumscribed, increased amount of normal pigment, differing from chloasma only in the peculiar form and size of the deposit.

Symptoms.—The number of "freckles" varies from a very few to immense numbers. They occur as brownish or yellowish-brown small, roundish, irregular spots, most commonly upon the face and hands. Rarely the number is very great, and they give to the skin an uncleanly appearance. They are apt to occur at all ages, but rarely before the third year. They are unattended with itching or other subjective symptoms.

Prognosis.—Usually favorable. Their course, when left to themselves, is chronic, lasting for years or a life-time. They ordinarily appear in the summer, fading away as cold weather approaches, to return the following summer.

Treatment.—The following application has usually been successful:

℞. Hydrargyri chlor. corrosiv.	gr. iij	0.2 Gm.
Acid. hydrochlorici dil	fʒ j	4. Cc.
Alchoholis	f℥ j	30. Cc.
Glycerini	f℥ ss	15. Cc.
Aquæ rosæ	ad f℥ iv	ad 120. Cc.

M. S.—Apply at bedtime, and remove with soap and water in the morning.

CHLOASMA.

Synonyms.—Liver spots; moth.

Definition.—A pigmentary disturbance of the skin, characterized by variously sized and shaped, more or less defined, smooth patches, or a yellowish, brownish, or blackish color.

Cause.—The affection may be idiopathic or symptomatic.

Idiopathic chloasma results from the irritation of long-continued scratching, such as occurs in severe eczema or pediculosis, the application of blisters and sinapisms, heat, the direct rays of the sun, and various medicinal and chemical substances.

Symptomatic chloasma occurs in connection with cancer, malaria, tuberculosis, disease of the suprarenal capsule (Addison's disease), disease of the womb, pregnancy (chloasma uterinum), neurotic disturbances, dementia, anemia, and chlorosis.

Pathology.—The affection consists of an increased deposit of the normal pigment in the mucous layer of the epidermis. The deposition of the additional pigment is the result of a nervous derangement, possibly of the trophic system.

Symptoms.—Chloasma is simply a discoloration of the skin, unattended by any alteration of the surface. The patches vary in size and shape; they may be as minute as a coin or as large as the hand, or much larger, even to a universal discoloration of the entire surface, and they may be roundish or irregular in outline. The usual color is yellowish, brownish, or muddy, or even blackish (*melasma melano-derma*).

In *Addison's disease,* of a typical character, "the coloration is brownish, with an olive-greenish or bronze tint, and is general, although, as a rule, especially pronounced upon regions having a disposition to normal increase of pigment, as the face, backs of the hands, axillæ, areolæ of the nipples, and the genital organs; the hair, also, may become darkened.

In *argyria,* or discoloration of the skin resulting from the internal use of nitrate of silver over a long period, the color is a bluish-gray, slate, bronze, or blackish, varying as to the shade. It occurs over the surface generally, but is more pronounced upon parts exposed, as the face and hands.

Chloasma uterinum occurs most frequently between the ages of twenty-five and fifty, seldom after the menopause, and is caused, in the greater number of instances, by changes, physiologic and pathologic, which take place in connection with the uterus. It is seen in the married and single, although more common in the former. Pregnancy is the most frequent cause, but it is also associated with either dysmenorrhea, chlorosis, anemia, and hysteria. It is seen in the mildest degree about the eyelids, especially during the menstrual epoch, as a duskiness or swarthiness of the complexion, either lasting a few days or being permanent. As usually encountered, however, chloasma of this variety consists in the presence of one or several patches, appearing generally about the forehead or other parts of the face, upon the trunk, about the nipples, and upon the abdomen. Rarely the entire face is covered with a discoloration, resembling a mask. Cases are recorded in which the pigmentary deposit was general, resembling Addison's disease.

Diagnosis.—Tinea versicolor and chloasma resemble each other in the color of the patches, but otherwise they have nothing in common. Tinea versicolor occurs on the trunk, while chloasma occurs upon the face and about the nipples, and in cases the result of pregnancy about the umbilicus, except in those comparatively rare instances in which the discoloration is diffused. The patches of chloasma are smooth, those of tinea versicolor furfuraceous, as can readily be demonstrated by gently scraping the discoloration with the finger-nail. The parasite is absent in chloasma.

Prognosis.—The outlook is favorable except in cases due to the prolonged use of silver nitrate, Addison's disease, tuberculosis, or cancer.

Treatment.—Except when due to organic disease or silver deposits in the skin, the pigmented areas may be temporarily removed by one of the following:

℞. Hydrargyri chloridi corrosiv.	gr. viiss	0.5 Gm.
Zinci sulphat.	ʒ ss	2. Gm.
Plumbi acetatis	ʒ ss	2. Gm.
Aquæ	f℥ iv	120. Cc.

M. S.— Lotion. Apply morning and evening. (Hardy.)

Or—

℞. Hydrargyri chloridi corrosiv.	gr. vj	0.4 Gm.
Acidi acetici dil.	fʒ ij	8. Cc.
Boracis	℈ ij	2.6 Gm.
Aquæ rosæ	f℥ iv	120. Cc.

M. S.—Lotion. Apply twice daily (Bulkley).

Or—

℞. Hydrarg. ammoniat.	ʒ j	4. Gm.
Bismuthi subnit.	ʒ j	4. Gm.
Petrolat.	℥ j	32. Gm.

M. S.—Apply frequently.

CALLOSITAS.

Synonyms.—Tyloma; callus; callosity.

Definition.—Callositas, or tyloma, consists in the development of a hard or horny, thickened patch of skin, variable in extent, of a grayish, yellowish, or brownish color, and unattended by pain. The most frequent location is upon the hands and feet.

Causes.—The principal cause is local pressure or friction, as in the case of the hands of the mechanic, the effect of his tools; or, if upon the foot, the result of ill-fitting shoes or from long marches. Callosities are also seen upon the fingers of violin, banjo, and harp players.

Pathology.—Hypertrophy of the horny layer of the skin is present, the corium remaining normal. The cells of the epidermis become so closely packed together as often to simulate horn-substance.

Symptoms.—Callositas consists in an increase in the thickness of the skin of the affected part, presenting a firm, dense, more or less circumscribed structure, the extent of hardness varying considerably. The patch of hardness is generally about the size of a coin, roundish in shape, and somewhat elevated above the surrounding skin. The color may be either grayish, yellowish, or brownish.

Callosities are usually situated upon the palms, fingers, soles, and toes, although other parts, if exposed to the cause, may also be the seat. At times great pain and discomfort are experienced from the growth.

Occasionally callosities are complicated by hyperemia, fissures, acute inflammation, abscess, erysipelas, and serve readily as foci for such cutaneous diseases as eczema and psoriasis. Their formation and development is always slow and gradual. If the cause be removed, the prognosis is favorable.

Treatment.—If the removal of the callous growth be desirable, the part should be repeatedly soaked in warm water, or a poultice applied, or warmed oil kept in contact by compresses of flannel, which will soften the induration and permit its removal by paring or scraping, layer by layer, with a sharp knife. Success has been obtained from the use of a plaster of india-rubber containing salicylic acid. Painting with diluted tincture of iodin once daily is often serviceable.

CLAVUS.

Synonym.—Corn.

Definition.—A corn is a small, circumscribed, usually flat, deep-seated hypertrophy of the epidermis, having a horny feel, projecting slightly from the skin, painful upon pressure, and situated for the most part about the toes.

Cause.—Continued pressure or friction, usually from ill-fitting or tight boots or shoes.

Pathology.—A clavus consists of a circumscribed, excessive hypertrophy of the epidermis, of the same character as occurs in callosity, and of a central portion—the *core*. The core extends deeply into the tissues, in the shape of an inverted cone, the base of the cone being directed outward and appearing upon the surface as a roundish elevation, its apex resting upon the papillary layer of the corium. The core of a clavus consists of a whitish, opaque, firm, tenacious body, composed of epidermic cells, arranged in concentric laminæ.

The pain attending the presence of corns results from pressure upon the true skin by the hard core, causing irritation of the nerve-filaments of the papillæ.

Corns existing between two toes are constantly bathed with the moisture of the part, which macerates and softens the formation, which thus receives the name of *soft corn*, in contradistinction to the hard corn.

Symptoms.—Until the growth attains a considerable size no discomfort, as a rule, is felt. After, however, its depth has reached the true skin, pain of an intermittent character, aggravated by pressure, is the chief symptom. Corns are often weather sensitive, being unusually painful before, during, or after the occurrence of storms, and should, therefore, not be confounded with gouty or rheumatic deposits below the skin.

Treatment.—If freedom from these annoying formations be desired, a properly fitting foot-covering must be worn. The pressure which results in the severe pain is limited by the use of the ringed protective plasters in common use.

To remove the corn, soaking with hot water, or a poultice kept in contact over night, will soften the part and permit of its ready removal with the knife.

For soft corns, the application of silver nitrate in solid stick form is highly spoken of, to be used after the growth has been sufficiently softened. The following application will usually remove the "corn":

℞.	Acidi salicylici ....ʒ iss	6. Gm.
	Ext. cannab. indicæ .....gr. x	0.6 Gm.
	Collodii ....f℥ j	30. Cc.

M. S.—Painted over corn at night and scraped off in the morning.

ICHTHYOSIS.

Synonyms.—Ichthyosis vera; fish-skin disease.

Definition.—Ichthyosis is a congenital, chronic deformity or hypertrophic disease of the skin, characterized by dryness, harshness, or general scaliness of the skin, or in the outgrowth of larger masses of a corneous consistency.

Varieties.—*Ichthyosis simplex; ichthyosis hystrix.*

Cause.—It is to be regarded as an affection which is born with the individual, although it does not usually manifest itself until after the first or second year of life. It is often hereditary.

Pathology.—"The diseased or, better, deformed skin is found micriscopically to be hypertrophied in various degrees, according to the development of the malady; the proliferation of its elements occurring in the connective-tissue, papillæ, stratum corneum, and blood-vessels. In well-marked cases of ichthyosis hystrix the elongated papillæ are surrounded by dense cones of the horny layer of the epidermis, more or less concentrically disposed, with sclerosis of the connective-tissue and a relatively unchanged rete. In this last particular the dense plaque of ichthyosis differs in texture from the wart" (Hyde).

Symptoms.—Ichthyosis displays wide variation in its symptoms. In one individual it amounts to slight inconvenience, while in another it may manifest itself in so pronounced a manner as to be the source of great discomfort and deformity. The two varieties named represent merely accentuated types of the disorder, rare in its fullest development, and, in the slightest, much more common than is generally believed.

A simple dryness and harshness of the skin, with only slight furfuraceous exfoliation, is termed *xeroderma*.

Ichthyosis simplex is the more common variety, consisting of a harsh, dry condition of the whole surface, accompanied by the production of variously sized and shaped reticulated scales, either small, thin, and furfuraceous, like bran, or large and thick, resembling fish-scales. Upon the extremities, the scales usually form diamond-shaped or polygonal plates, separated from one another by furrows or lines which extend down to the normal skin. In color, the scales are either whitish, grayish, or yellowish, and often have a silvery or glistening appearance. Rarely the color is olive-green or blackish (*ichthyosis nigricans*). The amount of scaling depends upon the age of the patient and the duration and severity of the disease.

Ichthyosis hystrix. With or without the development of the above variety, in this, the hypertrophy of the skin may occur in circumscribed patches or large areas, consisting of irregularly shaped verrucous, corneous, corrugated, wrinkled, or rugous masses, usually darker in color than those of the simple variety. They may occur upon the arms, as solid, warty patches, or upon the back, in the form of elongated, linear patches. They may constitute roughened, corrugated, papillary growths, or uneven, horny, blunt, or pointed, spinous, warty formations. In the latter case the elevations may reach several lines or more, and stand out from the skin like quills upon the back of a porcupine—hence the name hystrix. The amount and extent of the hypertrophy varies; the older the patient, the more highly developed it will usually be.

Course.—*Ichthyosis simplex* may involve the entire surface uniformly or appear more marked on the extremities, from the hips to the ankles and the arms and forearms. The affection is always worse in winter than in summer, the increased activity of the sweat glands at this season producing the most beneficial results. The course of the affection is essentially chronic, continuing throughout life, now better, now worse. Slight itching usually occurs.

Diagnosis.—The characteristics of the affection are so peculiar that an error in diagnosis is hardly possible. It is to be distinguished from the inflammatory affections of the skin which terminate in desquamation by the absence of any history of inflammation.

Prognosis.—While much can be done to alleviate the affection, the prognosis is unfavorable as regards permanent relief.

Treatment.—Local measures are alone of value for ichthyosis. The maceration of the accumulated masses of epithelial hypertrophy is accomplished by water-baths, either simple or medicated. The relief thus afforded the patient, while temporary, is comforting. Vapor and alkaline baths are also serviceable. Another valuable agent is soft soap in conjunction with baths, or alone, as a discutient. For severe cases, a "a sufficient quantity is to be rubbed into the skin twice daily for four or six days, during which period the patient is to refrain from bathing. A bath is first to be taken four or five days after the last rubbing, when, in fact, the epidermis has begun to peel off; afterward inunction with a simple ointment is to be applied, in order to prevent fissuring of the new skin."

The following is a useful formula:

℞.	Adeps benzoat.	℥ j	32. Gm.
	Glycerini	♏ xl	2.6 Cc.
	Petrolat.	℥ ss	16. Gm.

M. S.—Apply daily, after washing or bathing.

Or—

℞.	Potassii iodidi	gr. xx	1.3 Gm.
	Olei bubuli	f℥ ss	15. Cc.
	Adeps	℥ ss	16. Gm.
	Glycerini	fʒ ij	8. Cc.

M. S.—Apply after bathing (Milton).

VERRUCA.

Synonym.—Wart.

Definition.—A wart consists of a circumscribed hypertrophy of the papillary layer, with more or less epidermal accumulation, characterized by the appearance of a hard or soft, rounded, flat, or acuminated formation, or variable size.

Varieties.—The following varieties have chiefly a descriptive value: *verruca vulgaris; verruca plana; verruca filiformis; verruca digitata; verruca acuminata.*

Cause.—Obscure. Irritation, uncleanliness, and microörganisms are responsible for some forms.

Pathology.—While the anatomy of warts differs somewhat according to their variety, in all forms there exists as a basis of their formation a connective-tissue growth from which the papillary hypertrophy takes place. The interior of the growth is supplied by one or more vascular loops, from which their vitality is obtained.

Symptoms.—*Verruca vulgaris,* or the ordinary wart commonly seen on the hands, consists of a small, circumscribed, elevated growth, having a broad base seated securely upon the skin. Their consistency is either soft or firm, the surface smooth or rough, and the color that of the surrounding skin, or yellowish, brownish, or even blackish. They may develop upon any region of the body but are most commonly seen upon the hands and fingers.

Verruca plana differs from the vulgaris in being flat and broad in form, and but slightly raised above the level of the surrounding skin. Their most common location is either on the back or forehead.

Verruca filiformis assumes the shape of a minute, thin, conical, or thread-like formation, about an eighth of an inch in length. The most frequent location is the face, eyelids, and neck.

Verruca digitata consists of a slightly elevated, broad formation, about the size of a split pea, and marked by a number of digitations coming from its border, giving an appearance, in marked cases, resembling a crab. Their most frequent site is upon the scalp.

Verruca acuminata, known also as the pointed wart, the moist wart, the pointed condyloma, cauliflower excrescence, and venereal wart, consists of one or more groups of irregularly shaped elevations, often so closely packed together as to form a more or less solid mass of vegetations (*verrucæ vegetantes*). Their color depends somewhat upon the degree of vascularity, varying from a pinkish, bright red to a purple color. They occur, for the most part, about the genitalia of either sex. Upon the penis they usually spring from the glans and the inner surface of the prepuce. From the the inner surface of the labia and from the vagina in the female. They are also seen about the anus, mouth, axillæ, umbilicus, and toes. They may be either moist or dry, according to their location. About the genitalia, a yellowish, puriform secretion usually covers their surface, due to friction and maceration, which, owing to the heat of the parts, rapidly decomposes, producing a highly offensive, penetrating, and disgusting odor. Their size varies from that of a pea to that of an almond, an egg, or even the fist. Their development is rapid, attaining considerable size in a few weeks.

Prognosis.—Favorable.

Treatment.—For the smaller warts, excision by means of the knife or scissors afford the most satisfactory results. If the growth be large, and likely to be attended with considerable hemorrhage, as in cases of condyloma about the genitalia, the galvano-caustic wire or the Paquelin cautery are to be preferred. Transfixing the growth in several directions with long needles dipped in a fifty percent soltion of chromic acid has been recommended. The local application of caustics such as acetic acid, trichloracetic acid, nitric acid, nitrate of silver, or perchlorid of iron is often satisfactory. Painting of the growth with tincture of thuja occidentalis until their size is considerably reduced and then snipping them off with scissors is also a very efficient mode of treatment. The following applications are of value:

℞. Acidi salicylici	ʒ ss	2. Gm.
Ext. cannab. indicæ	gr. v-x	0.3-0.6 Gm.
Collodii	f℥ ss-j	15.-30. Cc.

M. S.—Apply once or twice daily.

Or—

℞. Acidi salicylici		
Acidi borici	āā gr. xv	āā 1. Gm.
Hydrargyri chlor. mitis ..	gr. x	0.6 Gm.

M. S.—Sprinkle over twice daily.

MOLLUSCUM EPITHELIALE.

Synonym.—Molluscum contagiosum; molluscum sebaceum.

Definition.—An infrequent epithelial affection characterized by the formation of discrete, pin-head to pea-sized, wax-like, whitish or pinkish elevations, the summits of which are flattened and have a central opening through which a cheesy fluid may be squeezed. It occurs usually in children and is slightly contagious. The lesions occur with greatest frequency on the eyelids and cheeks but may occur on the trunk. They grow very slowly and often disappear spontaneously by a process of sloughing but leaving behind no scar. Excision or cauterization may be performed for their removal. Ointment of ammoniated mercury is useful in slight cases.

COMEDO.

Synonyms.—Acne punctata nigra; black-heads or worms.

Definition.—A disorder of the sebaceous glands; characterized by the retention in the excretory ducts of an inspissated secretion which is visible upon the surface as yellowish or whitish pin-point and pin-head-sized elevations, containing in their centers blackish points.

Causes.—The exact etiology is unknown. Among the causes assigned are anemia, menstrual disorders, urethral irritations, dyspepsia, and constipation. Acne vulgaris is often associated with this condition.

Pathology.—Comedo is an affection of the sebaceous glands and ducts, consisting of an accumulation of sebum and epithelial cells in the glands and follicles, dilating the ducts to such an extent as to produce the point or elevation upon the surface. The obstructed gland may relieve itself, or it may continue distending until a papule is formed. The duct sometimes contains small hairs, and also the microscopic mite, *Demodex folliculorum,*—having a length of from 1-150 to 1-75 of an inch, and breadth of about 1-500 of an inch,—which was at one time supposed to be the cause of the affection.

Symptoms.—The affection is observed for the most part on the face, neck, chest, and back. Each elevation or blackhead or point is designated a *comedo;* if a number, *comedones.*

Each comedo is small, varying from a pin-point to a pin-head in size, having a brownish or blackish appearance, from the dust or dirt that has adhered to the unctuous surface. If they form in great numbers upon the face they are disfiguring, giving the individual the appearance of having had minute grains of powder implanted in the skin. There are no evidences of inflammation unless acne is associated, but, on the contrary, the skin has a dirty, greasy, unwashed appearance.

Diagnosis.—There is no condition resembling comedo, so that its recognition is easy, unless complicated with acne; but even then the inflammatory appearance of acne should prevent error.

Prognosis.—Favorable, although often remarkably obstinate.

Treatment.—Derangement of any of the functions of the body should be corrected, and strict attention be given to the rules for promoting the general health.

Local measures are usually sufficient. The parts should be thoroughly softened by bathing with soap and warm water, when the comedones are removed by friction with a Turkish towel, pressure between the thumb nails, the application of a watch-key, or the instrument known as the "comedo-extractor," and their return prevented by an ointment medicated, to meet the indications, with either sulphur, alkalies, or mercury.

Shoemaker recommends the following formula:

℞. Thymol	gr. x	0.65 Gm.
Acidi borici	ʒ ij	8. Gm.
Aquæ hamamel. Virg. dest.	fʒ iv	15. Cc.
Aquæ rosæ	f℥ j	30. Cc.

M. S.—Mop well over surface once or twice daily.

MILIUM.

Synonyms.—Grutum; tubercula miliaria or sebacea; acute punctata albida.

Definition.—An accumulation of sebum in the sebaceous glands that are minus their excretory ducts, characterized by the formation of small, roundish, whitish, sebaceous, non-inflammatory elevations, situated immediately beneath the epidermis.

Cause.—The origin of the affection is not understood.

Pathology.—The sebaceous gland is distended with the sebum, which is unable to escape, owing to the obilteration of the duct, nor can the contents be squeezed out, as no sign of aperture is to be found, the formation being completely enclosed. Rarely the retained secretion undergoes a metamorphosis into hard, calcareous, stone-like masses — sebaceous concretions or *cutaneous calculi*.

Symptoms.—Milia may occur upon any portion of the body; their usual seat, however, is upon the face, forehead, and about the eyes. They form gradually, are about the size of a millet seed, of a whitish, pearl, or yellowish color, hard, and of a rounded shape, giving the sensation to the touch of hard bodies embedded in the skin. They are not associated with inflammatory symptoms.

Diagnosis.—Milium and comedo are somewhat similar in appearance; the differences are that in milium the sebaceous gland is distended without an opening, while in comedo the duct of the gland is always patulous upon the surface. Milium usually exists singly, the skin looking normal; while comedo is more general, the surface having a soiled and greasy appearance.

Prognosis.—Favorable.

Treatment.—As a rule, no treatment is needed, the number being few and their presence of no consequence.

If their removal be desirable, two modes suggest themselves: one, to open the cyst with a fine-bladed bistoury, turning the contents out, and destroying the remaining sac by the application of either tincture of iodin or chromic acid; or the cyst may be destroyed by electrolysis.

SEBACEOUS CYST.

Synonyms.—Wen; sebaceous tumor; encysted tumor.

Definition.—A distention of the sebaceous gland and duct, with hypertrophy of the walls, forming a thick, tough sac or cyst, characterized by a firm or soft, more or less rounded tumor, having its seat in the skin or subcutaneous connective-tissue.

Cause.—Unknown.

Symptoms.—The development of wens is slow and insidious. The localities where they are most commonly observed are the scalp, face, back, and scrotum. The tumors occur singly or in numbers; in size from a pea to a walnut, or larger; in shape either rounded, flattened, or semiglobular; in consistency they are either hard or soft and doughy; they are freely movable and painless.

Treatment.—Excision, with careful and thorough dissection of the cyst is the only satisfactory mode of treatment.

KERATOSIS PILARIS.

A cutaneous affection characterized by pin-head sized papules situated at the mouths of the follicles resulting from epidermal accumulations or hypertrophy. The lesions are grayish, whitish, or blackish in color and are found most frequently on the extensor surfaces of the extremities. The skin is dry and rough but there is no itching. Infrequent bathing is believed to be the most common cause. Bathing with soft soap and alkaline water, followed by vigorous friction, and inunctions of petrolatum constitute the treatment.

HYPERTRICHOSIS.

Synonyms.—Hirsuties; hypertrophy of the hair; superfluous hair.

Definition.—A local or general overgrowth of the hair, either in normal or abnormal situations. When the growth occurs upon a mole it constitutes *nævus pilosus*. The cause of hypertrichosis is unknown. Some cases apparently arise from local irritation.

Treatment.—Removal of the hairs by means of electrolysis is the only satisfactory method of treatment. Shaving, extraction, and the use of depilatories are only of temporary value.

ELEPHANTIASIS.

Elephantiasis is the hypertrophic condition of the skin and underlying tissue, having its origin in lymphatic obstruction and characterized by edema, enlargement, thickening of the skin, overgrowth of the papillæ, and pigmentation. The causal lymphatic obstruction may be due to tumors, cicatrices, erysipelas, and the *Filaria sanguinis hominis.* It is most common in the tropics and is most often observed in male adults. The structural changes incident to the disease are hypertrophy of the entire skin and subcutaneous tissue, edema of affected structures, and dilatation of the blood-vessels and lymphatics with inflammation of the latter. The disease usually affects the leg and foot but the genitalia may be attacked.

The early stages of the disease consist of recurring attacks of an erysipelatoid inflammation. Restoration to normal is never complete and with each succeeding attack the part becomes larger. In a well-marked case, the enlargement is very great and the skin is thickened, pigmented, fissured and covered with papillomatous outgrowths. The affection is essentially chronic and pain is absent except in acute exacerbations. The fully-established disease is incurable but in the early stages, sedative applications, elastic compression, and mercurial inunctions, together with nutritious food, tonics, hygiene, etc., may arrest its progress. In marked enlargement resort to surgical means is necessary.

ONYCHAUXIS.

Hypertrophy of the nails. It may be congenital or acquired, idiopathic or symptomatic. Among the principal diseases to which it may be due, may be mentioned syphilis, psoriasis, leprosy, ringworm, ichthyosis, and neuritis. Traumatism may induce it. Treatment is unsatisfactory and very variable.

ATROPHIES OF THE SKIN.

ALBINISM.

Albinism consists in a congenital absence of pigment in the hair, skin, and eyes. The cause is unknown. It is believed to be influenced by heredity. In a typical case, the skin is unusually white; the hair is fine, silky, and whitish or yellowish-white in color; the pupils appear red; and the irides are lighter in color than normal. Sensitiveness to light, nicitation, nystagmus, high errors of refraction, and mental inferiority are rather common accompaniments. Partial albinism is common in negroes and such individuals are termed "piebald." Treatment is of no avail.

VITILIGO.

Synonym.—Leukoderma.

Definition.—An acquired condition characterized by areas devoid of pigment surrounded by hyperpigmented borders. It occurs usually in adult life and seems to be dependent upon some disturbance of innervation. Apart from this, the cause is obscure. The affected areas are attended by no changes other than loss of pigment which may also be absent from the hair in those regions. The pigment is increased on the borders of the patches. Its onset is slow and its course indefinite. It usually persists during the life-time of the individual. The treatment is unsatisfactory. Arsenic internally, and counterirritation to the patches may be tried.

SCLERODERMA.

Synonyms.—Sclerema; hidebound disease.

Definition.—A rare atrophic cutaneous affection, characterized by circumscribed or diffused induration, rigidity, and stiffening. It occurs usually in adult females. The direct cause is unknown. Exposure, rheumatism, neurotic disturbances, etc., may influence its production. The disease begins with stiffening and pigmentation of the integument. This increases and is followed by induration and rigidity. The skin meanwhile becoming atrophic, The surface of the skin is dry, smooth, and tense. In a marked case the joints also become fixed. In the early stage the skin is thickened but late in the disease it becomes thinned. The course is chronic and the prognosis is unfavorable. Massage and inunctions are of value in relieving the tension. Otherwise, treatment is of no avail.

MORPHEA.

Synonyms.—Addison's Keloid; circumscribed scleroderma.

Definition.—An atrophic disease of the skin, characterized by sharply circumscribed, firm, whitish-yellow patches, surrounded by a violaceous zone. The surface is smooth, shiny, and resistant. The lesion is most common on the trunk. The course is chronic.

Treatment.—Tonics should be administered internally, and massage, electricity, and the X-ray should be tried locally. The results of treatment, however, are not very encouraging.

CANITES.

Absence of pigment in the hair. It may be local or general, senile or premature. Premature whitening of the hair may be due to profound emotional disturbances, fright, shock, fear, worry, neuralgia, vitiligo, heredity, etc. It usually takes place slowly but may occur very suddenly. The treatment consists in the application of hair dyes. Internal medication is useless.

ATROPHY OF THE NAILS.

This may result from injury, disease of the nerves, syphilis, psoriasis, ringworm, etc., or it may be congenital. The nails become lusterless, brittle, and dwarfed. Treatment depends upon the cause but even in the most favorable cases some deformity remains.

ALOPECIA.

Synonyms.—Baldness; calvites.

Definition.—Partial or complete loss of hair. It may be congenital or acquired. The acquired form may be senile or premature, idiopathic or symptomatic. The idiopathic variety occurs without obvious internal or external causes and is seldom amenable to any form of treatment. Symptomatic alopecia is that form of the affection which results from syphilis, infectious fevers, seborrhea, lupus erythematosus, parasitic diseases of the skin, psoriasis, eczema, and similar conditions.

The prognosis depends on the cause. In congenital, senile, and idiopathic alopecia the hair seldom regenerates. In symptomatic alopecia, the possibility of return of the hair is directly proportionate to the removability of the cause.

Treatment.—This varies with the underlying cause. Internally, tonics, especially strychnin, iron, and arsenic, together with fluid extract of jaborandi, 10 ♏ (0.65 Cc.), three times daily should be administered. Locally, stimulating applications should be made varying with the character of the local cause. Seborrhea being a very frequent cause, its treatment is applicable in most cases.

ALOPECIA AREATA.

Definition.—Baldness in circumscribed areas. These areas occasionally coalesce, producing *alopecia universalis.* Most cases are due to some disturbance of the nervous system while others seem to owe their origin to a parasite. The condition is one of atrophy and affects the entire hair and the adjacent skin. The most common situations for the disease are the scalp, beard, eyebrows, and eyelashes, but in very rare instances the pubic and axillary hair may also be lost. As ordinarily observed, the disease presents one or more rounded, circumscribed, smooth, white patches of baldness. There are no prominent follicles or broken-off hairs as in ringworm. The skin may at first be somewhat inflammatory but soon becomes pale and atrophic. The onset may be sudden or gradual and the duration is indefinite.

Prognosis.—The course is rather chronic. Recovery is the rule in children but usually requires a period of several months. In older persons, the prognosis should be guarded; the hair returns but requires a much longer period. In middle-aged or older, adults, the outlook for the return of the hair is unfavorable. Relapses are common. The return of the hair is evidenced first by the appearance of fine, white, downy hairs over the affected areas; these later become converted into or are replaced by the natural hair.

Treatment.—Tonics should be administered internally. Arsenical preparations are especially valuable. Fluid extract of jaborandi, ♏x (0.65 Cc.), is very beneficial in some cases. Locally, stimulating applications should be employed. The following is an example:

℞. Betanaphthol ....ʒ i	4. Gm.
Petrolat. ....℥ i	32. Gm.

M. S.—Apply locally twice daily.

NEW GROWTHS OF THE SKIN.

KELOID.

Synonyms.—Cheloid; keloid of Alibert.

Definition.—An abnormal growth of connective-tissue developed at the site of an injury. It is observed most frequently in negroes and usually follows lacerations, burns, bites, and destructive lesions. Occasionally it arises spontaneously. In the early stages, the growth appears as a small, pale-red nodule but as it progresses it increases in size, sending out claw-like processes. It is smooth, dense, and of a pinkish color but may be darkly pigmented. Subjective symptoms are absent. The affection usually occurs over the sternum.

Treatment.—Excision should never be performed as the resulting scar will give rise to greater deformity than the keloid. Multiple scarification, electrolysis, mercurial plaster, and the X-ray may be employed. In most cases the growth is permanent.

XANTHOMA.

Synonyms.—Xanthelasma; vitiligoidea.

Definition.—An abnormal cutaneous condition characterized by the formation of circumscribed flat or tubercular yellowish patches. The flat variety, *xanthoma planum*, is most often observed on the eyelids and consists of smooth, soft, sharply circumscribed, buff-colored, slightly elevated patches. The tubercular form, *xanthoma* *tuberosum,* occurs elsewhere on the body as variously sized, smooth elastic yellow nodules.

Causes.—Frequently no cause can be detected. Female sex, middle life, jaundice, and diabetes are factors in some cases.

Treatment.—Usually no treatment is necessary, as the growths usually remain stationary. Removal may be accomplished by electrolysis if necessary. Excision or the galvanocautery are seldom necessary.

LUPUS ERYTHEMATOSUS.

Synonyms.—Seborrhea congestiva; lupus non-exedens; lupus erythematodes; lupus sebaceus.

Definition.—A chronic, superficial, new-growth formation of the skin characterized by sharply circumscribed reddish patches covered with adherent grayish or yellowish scales.

Causes.—The etiology is obscure. The disease is observed with greatest frequency in the female sex during early and middle adult life. Many cases are preceded by local congestive disorders such as acne rosacea, seborrhea, eczema seborrhoicum, sunburn, chilblains, etc. General ill health is also an etiologic factor. By many observers, the affection is believed to be an expression of tuberculosis.

Pathology.—The true nature of the affection has not been as yet definitely determined. Many observers believe it to be a new growth while others view it as a chronic inflammation. The earliest change is believed to be capillary obstruction. The principal structural alterations of the disease are to be found in the corium, consisting largely of a growth of reticulated, adenoid tissue, associated with perivascular infiltration. The excretory parts of the glandular structures are to some extent infiltrated. Edema of the prickle cells and cutis is also present. The scarring may result from degeneration of the sebaceous glands or the elastic fibers. The epidermis eventually becomes atrophic. The affection is believed to result from the toxin of tuberculosis, but the lesion possesses none of the characteristics of tubercular growths.

Symptoms.—The disease presents itself in four clinical varieties:—circumscribed, diffuse, telangiectatic, and nodular. The circumscribed form is perhaps the more common and is the variety usually observed on the nose, cheeks, ears, and scalp. Attention is first called to the condition by the presence of one or more pin-head to pea-sized, scaly, reddish spots, the borders of which may be elevated. They grow slowly, as a rule, and after a certain size is attained they may remain stationary or coalesce forming large patches. These patches are well-defined and sharply marginated, being separated from the healthy integument by an elevated border. The surface of the lesion is covered with scanty grayish scales which are firmly adherent and project into the follicular openings. The central portion of the disease is slightly depressed and atrophic and the ducts of the sebaceous glands are distended and patulous. The color of the patch is pinkish or reddish with a violaceous tinge, most marked at the border. On taking the affected skin between the fingers it is found to be infiltrated and thickened. Mild itching and burning are present. A common situation for the disease is the face, involving both cheeks and the nose at the same time and presenting the appearance of a butterfly with outstretched wings. Less frequently the ears, scalp, hands, and mucous membranes may be attacked. The lesions are usually symmetrically distributed. The course of the disease is essentially chronic. Involution occasionally occurs spontaneously but malignant changes are very rare.

Diagnosis.—Lupus erythematosus may be distinguished from other affections, especially lupus vulgaris, by its occurrence in adult life, its slow course, its symmetric distribution, the superficial character of the sharply-defined scaly patches with distended glandular openings, the atrophic scarring, and the absence of ulceration or nodules.

Prognosis.—The course of the disease is very chronic and extends over several years. Many cases never show any improvement. A few undergo spontaneous involution; and a limited number respond to treatment. The prognosis should always be guarded.

Treatment.—Internal treatment has little or no effect on the disease except in those instances in which definite internal affections exist, under which circumstances internal medication is *indirectly* of value.

Locally, moderately stimulating applications are of most benefit. A common practice is to wash the face nightly with soap (green soap if the patch appears sluggish) and apply some preparation of sulphur such as—

℞.	Sulph. precip.	ʒ i	4. Gm.
	Petrolat.	℥ i	32. Gm.

M. S.—Apply locally.

Or—

℞.	Zinc sulphat.		
	Potass. sulphid.	āā ʒ i	4. Gm.
	Aquæ	f℥ iv	120. Cc.

(Dissolve separately and then mix.)

S.—"Lotio Alba"—apply locally.

If much roughness or irritability results:—

℞.	Sulph. precip.		
	Acid. salicyl.	āā gr. x	0.6 Gm.
	Ung. aquæ rosæ	℥ i	32. Gm.

M. S.—Apply locally (Stelwagon).

Unna applies a paint consisting of ten parts of collodion and one to two parts of green soap to which three to five percent of salicylic acid may be added to increase its activity. The application of mercurial plaster and the painting of the lesion with liquor potassa, carbolic acid, salicylic acid in collodion, resorcin in collodion, and silver nitrate deserve passing mention. Strong caustics are occasionally employed but the scarring they induce is a disadvantage. Among other measures useful at times may be mentioned freezing with ethyl chlorid, scarification, curetting, electrolysis, galvanocauterization, phototherapy, and radiotherapy.

LUPUS VULGARIS.

Synonyms.—Lupus exulcerans; lupus exedens; lupus vorax.

Definition.—A neoplastic cellular infiltration caused by the tubercle bacillus, producing papules, nodules, and patches which either ulcerate or atrophy, leaving scars (Crocker).

Cause.—The direct cause is the *tubercle bacillus*. The disease usually begins in the first or second decade of life and is never congenital. In many cases there is an hereditary predisposition.

Some cases result from local inoculation. Evidences of tuberculosis elsewhere are often present.

Pathology.—The process consists essentially of a small round cell infiltration beginning in the corium and gradually invading the other layers. Circumscribed areas are encountered which possess all the structural characteristics of miliary tubercles (epithelioid cells, giant cells, etc.). Tubercle bacilli may be demonstrated but are very scant. Necrotic degeneration takes place in these areas followed by proliferation of the connective-tissue cells and the production of scar-tissue.

Symptoms.—The disease usually begins on the face as one or more deep-seated, pin-point to pin-head dull red spots. These gradually develop into small, semitranslucent brown nodules ("Apple-jelly nodules of Jonathan Hutchinson"). As the disease progresses, these nodules extend and eventually coalesce forming dull red, soft, elevated patches with firm, more or less nodular borders. More or less scaliness may be present. The nodules may remain stationary for a variable period but always terminate either in ulceration with scar formation or in absorption. A fully-developed patch of lupus tissue shows the presence of papules, nodules, flat infiltrations, ulceration, scar formation, and atrophic areas in varying degrees. At times papillomatous outgrowths may be found on the border. Frequently the lesion shows retrograde changes at one side and on the other, evidences of advancement. Slight pain may be present. The course is very chronic.

Diagnosis.—The characteristic features of this disease that serve to distinguish it from syphilis, epithelioma, and other ulcerative affections are: the beginning early in life, the slow course, and the superficial ulceration, together with papules, semitranslucent nodules, flat infiltrations, and scarring. The ulcers are multiple, have soft undermined edges, and give rise to little or no pain.

Prognosis.—In cases in which the lesions are small and the patient is young, cure may be effected by appropriate treatment. Usually the disease is refractory to treatment, and when one patch is destroyed another makes its appearance. The course is essentially chronic and the duration indefinite. The possibility of systemic infection should be borne in mind.

Treatment.—In all cases, the patient should receive the treatment recommended for tuberculosis in general, in addition to the various local measures for the diseased integument.

Locally, perhaps the most beneficial with the least deleterious results is the X-ray treatment. Phototherapy, after the method of Finsen is of value but requires a long period of treatment to be of value. Radium has also been reputed to be of benefit but observations have been few in this direction. Extirpation of the diseased structure has been practiced extensively. This may be accomplished by curetting, cauterization, electrolysis, or excision. The caustics most commonly employed for this purpose are pyrogallic acid (20 percent plaster), arsenious acid (30 percent paste), and chlorid of zinc. The galvanocautery and Paquelin cautery are also used. Scarification is also a useful method of treatment in some cases.

Frequently when first encountered, the lesion is in an irritable state either as the result of previous treatment or of unknown causes. In such cases, soothing applications are of benefit. Calamin lotion, diachylon ointment, and the ointment of the oleate of mercury (10 percent), one dram to the ounce of ointment base. Brooke advises the following:

℞.	Hydrarg. oleat (5 per cent.)	℥ i	32. Gm.
	Pulv. zinci oxidi		
	Pulv. amyli	āā ʒ ii	8. Gm.
	Vaselin	ʒ iv	16. Gm.
	Acid. salicyl.	gr. xx	1.3 Gm.
	Ichthyol	♏xx	1.3 Gm.

M. S.—Apply locally twice daily.

Plasters are often of value, particularly mercurial plaster, salicylic acid plaster (20 percent), and resorcin plaster. Various other modes of treatment may be employed according to the indications.

SCROFULODERMA.

Definition.—A tuberculous condition of the skin occurring in strumous individuals characterized by ulceration and associated usually with suppurating lymphatic glands. The disease begins in the lymphatic glands which undergo necrosis discharging through the overlying skin. The ulceration in the skin is violaceous in color and has thin undermined edges, its base being made up of pale granulations. These ulcers may occur anywhere on the body but are most common on the neck. They spread slowly and sometimes show a tendency to heal and form connective-tissue. Other manifestations of the strumous diathesis such as otorrhea, ocular inflammations, lymphatic enlargements, etc., are often present.

Treatment.—The general health should receive careful attention. Cod-liver oil, syrup of the iodid of iron, hydriodic acid, quinin, and similar drugs should be administered. Fresh air, sunlight, exercise, bathing, nutritious food, and other similar measures should be prescribed.

Locally, salicylic acid (gr. x) in lead plaster (50 percent) is a very efficient application. The oleate of mercury, and boric acid may also be used. The most efficient method of treatment is extirpation and this may be accomplished by curetting, excision, or the use of caustics such as pyrogallol.

SYPHILIS CUTANEA.

Synonyms.—Pox; lues; syphiloderma.

Definition.—A chronic, specific, contagious, hereditary disease characterized for the most part by lesions on the skin but may affect any tissue of the body. It is caused by contact with some special infecting agent as yet unknown.

The period of incubation between the time of exposure and the appearance of the first manifestation is about three weeks. This initial manifestation may appear as a scaly papule, superficial erosion with an indurated base, or the ordinary chancer. Within a month or six weeks after the appearance of the initial lesion the characteristic cutaneous eruption becomes manifest.

Symptoms.—The eruption of syphilis consists of nearly every form of primary lesions occurring on the skin, including macules, papules, pustules, tubercles, vesicles, blebs, and ulcerations (gummas). They may be conveniently divided into those of the secondary and those of the tertiary period.

The secondary eruptions include the macular, papular, pustular, bullous, and in very rare instances vesicular manifestations. They appear usually within four to six weeks after the initial lesion. They are generalized, symmetric, and copper-colored and as a rule devoid of itching. In negroes, itching is often present. The presence of several types of lesions in various stages of development (polymorphism) and the tendency to gyrate configuration are characteristics of this disease. Associated with these dermal manifestations are headache, malaise, fever, muscular and osseous pains, enlargement of the glands, sore throat, loss of hair, anemia, and failing health. Pigmentation often follows the disappearance of these lesions and lasts a considerable period.

The tertiary eruptions include the tubercular and gummatous lesions. These are circumscribed, non-inflammatory, and assymmetric. They are deep-seated and tend to destructive changes.

The Macular syphiloderm or roseola is characterized by the appearance of variously sized and shaped ill-defined macules, at first rose-red but later brownish, most marked in the trunk. The face is free in most cases. The lesions consist of hyperemia with slight cellular infiltration in contradistinction to the other lesions in which there is a dense, circumscribed, round-cell infiltration.

Diagnosis.—The history, the absence of catarrhal symptoms, the presence of the enlarged glands and induration of the chancer, loss of hair, crescentic arrangement of the eruption, the absence of itching, the presence of moist papules, and the color of the lesions will serve to distinguish this condition from tinea versicolor, measles, and eruptions resulting from the ingestion of drugs and certain kinds of food.

The Papular syphiloderm may appear as the large or small papular eruption and may occur either as an early or late manifestation. The large papular syphilide is characterized by sharply defined, flat, brownish-red lesions varying in size from an eighth to a half an inch in diameter. They are widely distributed, occurring with greatest frequency on the forehead, back of the neck, chin, arms, legs, and genitalia. When occurring on mucous surfaces or on opposing skin surfaces they are transformed into moist papules or "mucous patches." Moist papules may become the seat of vegetative growths. When the large, dry papular syphiloderm desquamates the eruption is termed papulosquamous.

Such eruptions are most commonly encountered on the palms of the hands and soles of the feet. The small papular syphiloderm consists of a profuse eruption of pinhead to millet-seed-sized, round or acuminate papules most marked on the trunk, arms, and legs. They possess the grouping and color characteristic of syphilitic eruptions in general.

Diagnosis.—While the eruption for the most part is papular, other types are usually present coincidently and this polymorphism, together with the history, distribution grouping, absence of subjective symptoms, and the presence of concomitant signs of syphilis serve to distinguish this form of the disease from psoriasis, lichen ruber, lichen scrofulosorum, keratosis pilaris, and other affections.

The Vesicular syphiloderm is a rare type of the disease. It consists of small umbilicated vesicles occurring when the skin is thin. Many of these become pustules before being detected. Other lesions are present at the same time, thus distinguishing it from eczema, herpes zoster, herpes simplex, dermatitis herpetiformis, etc.

The Pustular syphiloderm presents several features worthy of passing mention. In some cases the lesion is small and acuminated (*miliary pustular syphiloderm*) varying in size from a pinhead to a millet-seed and situated on a dull red base. The eruption is widely distributed and may be grouped or disseminated. The pustules are situated at the openings of the follicles. The pustules in other cases are large and acuminated resembling the lesions of acne and small-pox (*varioliformis syphilide*). Such lesions are most frequently found in the scalp, face, and trunk and are accompaniel by superficial ulceration. The pustules in syphilis are sometimes small and flat, resembling impetigo (*impetiginous syphilis*). The lesions of this form seldom exceed the size of a pea and show a marked tendency to arrange themselves in irregular groups. They are most often situated on the nose, around the mouth, and in the hairy regions.

They rupture early, giving rise to yellowish, greenish, or brownish crusting, on removal of which ulceration is exposed to view. Another variety of pustular syphilis is that in which the lesions are large and flat, resembling ecthyma. These pustules are about the size of the fingernail and are situated upon a dark red base. Crusting is profuse and beneath it there is ulceration. It occurs usually upon the back, shoulders, and extremities. *Rupia* is a malignant form of this large, flat pustular syphiloderm characterized by abundant greenish, blackish crusts arranged in concentric strata beneath which deep ulceration is taking place.

Diagnosis.—It is very important to distinguish this form from the affections it resembles for obvious reasons. In *acne* there are no concomitant signs of syphilis, the affection is chronic, the lesions consist of papules, pustules, and tubercles, and ulceration is absent. In *small-pox* there are severe constitutional symptoms of a different nature from those in syphilis, the lesions are first papular with shot-like induration, then vesicular with umbilication, and finally pustular, and the concomitant signs of syphilis are absent.

The Tubercular syphiloderm occurs as a manifestation of the tertiary stage of the disease and is characterized by reddish-brown, smooth, deep-seated nodules, varying in size from a pea to a hazel nut, and arranged in incomplete circles, upon the coalescence of which serpiginous patches are often formed (serpiginous tubercular syphiloderm). Occasionally these lesions are disseminated. The termination of this form of syphilis is either disappearance by absorption followed by pigmentation or by ulceration and scarring.

Diagnosis.—The tubercular syphilide may be mistaken for lupus vulgaris but it begins at a much later age, has an entirely different history, the concomitant signs of syphilis may often be detected, and the course is more rapid. In syphilis the nodules are hard, the ulcers are deep-seated and have sharp cut edges, and profuse discharge and crusting, and the resultant scars are smooth, soft, and white. Syphilis responds to treatment with mercury and the iodids.

The Gummatous syphiloderm is the late tertiary manifestation and consists of a sharply-defined infiltration of the skin and subcutaneous tissue which appears as one or more rounded, painless, non-inflammatory tumors. It begins very small and slowly increases until it reaches about the size of a walnut, when it suppurates and discharges through the skin, leaving behind a clear cut, deep ulcer. The resultant scar is white and smooth and somewhat depressed. The history, course, and character of the ulcer separate it from other ulcerating conditions.

The Bullous syphiloderm occurs most often in children as a manifestation of congenital syphilis (*pemphigoid syphilide*) but also late in the acquired variety of the disease in individuals in poor health. The blebs are discrete and their contents are at first serous and later purulent. As they dry they form dark crusts beneath which ulceration takes place.

Hereditary syphilis presents all the cutaneous eruptions common to the acquired form but lacks the initial lesion. The skin manifestations appear usually within the first six or eight weeks, the most frequent of which are the macular, papular, and bullous. The macular eruption consists of copper-colored spots and patches situated on the palms, soles, genitalia, and buttocks, associated with an apparent eczema intertrigo and a few moist papules. The papular variety may coexist with the macular and present the usual characteristics. The bullous form of the eruption is the most serious type of hereditary syphilis. The blebs are usually present at birth and are observed in most cases on the soles, palms, and on the toes and fingers, sometimes interspersed with macules and papules. The bullæ are discrete, flaccid, and surrounded by a brownish ring of infiltration. The base is ulcerated or excoriated. The concomitant signs in hereditary syphilis include constant coryza ("sniffles"), emaciation, apparent senility, mucous patches, Hutchinson's teeth, and dactylitis.

Prognosis.—In most cases in which the treatment can be properly carried out the prognosis as to cure is favorable. A very few cases progress toward a fatal termination in spite of treatment. Partially treated cases are liable to be masked and the disease manifest itself internally. In infants, the outlook is uncertain. In all cases, the prognosis depends on the general health of the patient, the severity of the attack, and whether the appropriate treatment can be instituted.

Treatment.—The indications are to restore the patient's general health by the use of iron, quinin, strychnin, etc., and combat the disease by mercury and the iodids. In the early secondary eruptions various preparations of mercury, particularly the protiodid, the bichlorid, the biniodid, mercury and chalk, and mercurial ointment in inunctions are indicated, being careful to prevent ptyalism. In hereditary syphilis, mercurial inunctions are of most value. In the tubercular and gummatous lesions of the skin potassium or sodium iodid in from five to ten grain doses is the drug to be employed. Larger doses than these are usually unnecessary. In the intermediary stages both mercury and potassium iodid should be administered. Usually the mercurial medication is given by the mouth, but inunctions, subcutaneous and intravenous injections, and fumigations are also employed. No matter what method is employed the mercurial should be given continuously for a year to eighteen months and followed by potassium iodid for six months or a year according to the symptoms.

Locally, applications are usually made only when the lesions are moist or ulcerative. In diffuse macular and papular eruptions a bichlorid of mercury bath (1 to 5000) or dusting with calomel may be prescribed. For moist papules calomel dusting powder is indicated. In tubercular or gummatous lesions, ammoniated mercury and mercurial plaster are of value. In offensive ulcerations potassium permanganate, bichlorid of mercury, black wash, etc., should be employed.

LEPROSY.

Leprosy is an endemic, chronic, infectious disease due to the *lepra bacillus* characterized by lesions in the skin and nervous system particularly, consisting for the most part of tubercular infiltration. Any structure may be attacked. It may be preceded for an indefinite period by various premonitory symptoms such as malaise, loss of appetite, mental depression, febrile paroxysms, etc. The manifestations of the disease include disturbances of sensation (anesthesia), pigmentation, tubercles, accentuation of the normal folds of the skin, ulceration, and deformities. The cause is unknown. The prognosis is unfavorable and the treatment is unsatisfactory.

DISORDERS OF SECRETION.

HYPERIDROSIS.

Synonyms.—Hydrosis; ephidrosis; idrosis.

Definition.—A disorder of the sweat glands, characterized by an increased secretion of sweat. The sweating may be either general or partial, but unilateral sweating, or hyperidrosis, is the condition here considered.

Causes.—Unknown. It may be inherited. Disorders of the sympathetic nervous system give rise to it in many instances. The condition is purely functional in character.

Symptoms.—The affection may be unilateral or bilateral, local or general, acute or chronic, and constant or paroxysmal. The quantity of secretion may be comparatively small or very large.

Local hyperidrosis occurs most commonly upon the palms, soles, axillæ, and genitalia.

Hyperidrosis of the palms may be so profuse that the fluid accumulates and keeps the parts constantly macerated, the wearing of gloves being impossible, for as soon as the parts are wiped dry they are again bathed in the secretion. Jamieson states that hyperidrosis of the hands is very common in those who are daily excessive spirit drinkers.

Hyperidrosis of the soles is a disagreeable and often distressing condition, as the socks and shoes become saturated, and thus keep the soles constantly bathed, allowing the macerated epidermis to peel off, leaving a more tender skin exposed, causing pain and distress when walking. The maceration of the epidermis, and the secretion about the toes, together with the moisture of the socks and the soles of the shoes, produce a most disagreeable, disgusting, and persistent odor, which is termed *bromidrosis pedum*.

Hyperidrosis of the genitalia attacks males more particularly, giving rise to a disagreeable, penetrating odor.

Bromidrosis is the designation when the secretion has an offensive odor.

Chromidrosis is the designation when the fluid poured forth is variously colored.

Uridrosis is the designation when the excretion from the sweat-glands contains the elements of the urine, and particularly urea.

Phosphoridrosis is the designation when the perspiration appears luminous in the dark.

Hematidrosis is the designation when the sweat contains blood.

Prognosis.—The majority of cases are extremely intractable, but in local hyperidrosis, particularly of the feet, the prognosis is favorable. Relapses may occur.

Treatment.—If the sweating is generalized, a careful search should be made to determine the underlying systemic cause and the internal treatment should be governed accordingly. Atropin sulphate, gr. 1-20 - 1-60 (0.00054-0.001 Gm.), twice daily, ergot in pill or solution, agaracin, gr. 1/6 (0.011 Gm.), gallic acid, quinin, mineral tonics, and sulphur, ʒ i (4. Gm.), twice daily, have been highly recommended for this condition.

Local treatment, however, is more efficacious. The parts should be cleansed and immediately dried, and then dusted with some one of the numerous dusting powders. The following is a valuable powder:

℞.	Acidi salicylici	gr. xx	1.3 Gm.
	Zinci oleat.	℥ j	32. Gm.

M. S.—Use locally.

Perhaps the very best local application is tincture of belladonna either diluted or full strength. Aristol as a dusting powder is very satisfactory.

In hyperidrosis of the palms and soles, the following are valuable, first washing the parts with a weak solution of carbolic acid:

℞.	Acidi salicylici	ʒ ss	2. Gm.
	Cretæ præp.	℥ j	32. Gm.
	Aluminis exsic.	℥ j	32. Gm.

M.—et powder finely.
S.—Apply to parts with puff-ball.

Or—

℞.	Acid. salicylici	3 parts.
	Pulv. amyli	10 parts.
	Pulv. soapstone	87 parts.

M. S.—Sift into shoes and stockings.

Or—

℞.	Sulphur. lotum.	gr. xxx	2. Gm.
	Pulv. arrowroot	ʒ iv	16. Gm.
	Acid. salicylici	gr. vij	0.45 Gm.

M. S.—Dust over feet and between toes.

Or—

℞.	Potassii permanganat.	gr. ij	0.13 Gm.
	Aquæ destil.	f℥ j	30. Cc.

M. S.—Apply locally.

A saturated solution of boric acid, alone or in powder, with equal parts of acetanilid, applied frequently to the hands and feet, often proves curative.

For obstinate cases, involving the palms or soles, the following plan of treatment, as suggested by Hebra, will be found of the greatest service. It is imperative that the various steps be closely followed:

"The parts are to be cleansed with water and soap, and the following ointment applied on pieces of cloth cut to the size of the region. Lint smeared with the ointment is also to be placed between the toes or fingers, so that every portion of the skin may be covered with a layer of the ointment.

℞.	Emplast. diachyli	℥ iv	120. Gm.
	Olei olivæ	f℥ iv	120. Cc.

The plaster to be melted and the oil added and stirred until a homogeneous mass results.

S.—To be used on cloths.

"The clothes are to be changed every twelve hours, when the parts are not to be washed, but rubbed with dry lint and starch dusting powder, after which new dressings are again to be applied in the same manner. This proceeding is to be continued from one to two weeks. When the disease is upon the soles, the patient may walk about in loose shoes." After a week or ten days the ointment may be discontinued, but the dusting powder is to be used for a considerable period. If relapses occur, the original treatment should again be instituted.

Painting the soles and under and between the toes with a one percent solution of formalin, morning, noon, and night, has given good results in a number of instances. A few drops of the solution may be put in the boot or shoe.

Among other methods of treatment may be mentioned the application of a one percent alcoholic solution of quinin, the use of astringent lotions containing alum, tannic acid, and similar substances (ʒ i-viii to the pint of water), the dusting of tartaric acid on the parts when there are no abrasions, and the employment of electricity.

ANIDROSIS.

Definition.—A functional disorder of the sweat-glands, characterized by a diminished or insufficient secretion of sweat.

Causes.—Anidrosis may be due to a congenital deficiency of the sweat glands or it may result from injury to a nerve, during the course of chronic diseases of the skin, as ichthyosis, eczema, psoriasis, lepra, and elephantiasis arabum. In rare cases an individual ceases to sweat entirely at times; in such cases the general health is impaired, and during the hot season much suffering may result.

Treatment.—The activity of the skin and sweat glands should be promoted by the ingestion of large quantities of water, hot baths, steam baths, friction, electricity, and the use of sudorifics, especially pilocarpin. In congenital cases, the treatment is of no benefit. The harshness and dryness of the skin in such cases may be relieved to some extent by oily applications.

SUDAMINA.

Synonyms.—Sudamen; miliaria crystallina (Hebra).

Definition.—A non-inflammatory affection of the sweat-glands; characterized by the rapid development of millet-seed-sized, translucent, whitish vesicles in great numbers upon any portion of the body.

Cause.—A high bodily temperature, causing unusual activity of the sudoriparous glands. The affection is common in febrile diseases.

Pathology.—The glands being excited beyond their capacity for normal excretion, the excessive fluid, instead of escaping upon the surface, collects between the layers of the epidermis, in the form of minute, translucent pin-point-sized vesicles.

Symptoms.—An ephemeral rash. Each minute vesicle is distinct, but they exist in great numbers, very closely resembling drops of free sweat. They develop rapidly, never coalesce, become puriform, or rupture. Fresh crops form from time to time. Their duration is transitory; the fluid is absorbed, the covering of each dries, forming a thin, delicate membrane, which disappears as a slight desquamation.

Treatment.—The treatment is that of the disease with which they occur.

MILIARIA.

Synonyms.—Lichen tropicus; miliaria rubra; miliaria alba; prickly heat.

Definition.—An acute inflammation of the sweat glands, characterized by the development of discrete, whitish or reddish, pin-point and millet-seed-sized papules, vesicles, or vesiculopapules, productive of prickling, tingling, and burning sensations of a most aggravated character, associated with more or less malaise.

Causes.—Excessive heat, the result of excessive or tightly-fitting clothing, or a high external temperature is the exciting cause. The affection is most frequent in fleshy adults who perspire freely, and in children. Nervous prostration; severe dyspepsia and general debility seem to predispose to "prickly heat."

Varieties.—*Miliaria papulosa; miliaria vesiculosa.*

Pathology.—The pathology of the two varieties is the same—both inflammatory affections of the sweat glands; in the one papules, in the other vesicles, develop about the orifices of the excretory ducts.

In either variety occurs hyperemia of the vascular plexus of the sweat glands, followed by slight exudations about the ducts, giving rise to the minute papules or vesicles, which remain until the cause has been modified or removed, when they are rapidly absorbed.

Symptoms.—*Miliaria papulosa*, known as lichen tropicus and "prickly heat," is of sudden onset, with the occurrence of numerous minute, acuminated bright-red papules, about the size of a pin-head or millet-seed, and but slightly raised above the level of the skin. The papules are preceded by and accompanied with sweating (hyperidrosis) and distressing, tingling, pricking, and burning sensations. If the attack be severe, vesicopapules and vesicles are freely interspersed among the numerous papules. Rarely the secretion of sweat is notably diminished.

Miliaria vesiculosa; in this variety, instead of papules, immense numbers of vesicles develop, of the size of pin-points and pinheads, of a whitish (*miliaria alba*) or yellowish-white color. The surface from which they arise is of a bright-red color, owing to each vesicle being surrounded by an areola (*miliaria rubra*). The vesicles are preceded and accompanied by sweating (hyperidrosis) and most distressing tingling, pricking, and burning sensations.

Either variety may attack all parts of the body, but the abdomen, chest, back, neck, and arms are regions usually invaded.

Duration.—This varies with the cause. It may appear, fully develop, and disappear in a few hours. In those predisposed it may continue more or less marked throughout the entire summer.

Diagnosis.—If the cause, nature, and seat of the affection are taken into consideration, no error should occur.

Eczema papulosum has a resemblance to "prickly heat," but the course of eczema is slow, and the papules are larger, more elevated, and firmer than those of miliaria papulosa.

Eczema vesiculosum and miliaria vesiculosa are to be differentiated by the marked differences in the progress of each,—the former slow, the later rapid; the vesicles of the former rupturing spontaneously, those of the latter only when severely irritated.

Sudamen is not an inflammatory affection, while miliaria is.

Prognosis.—The affection is often most rebellious in fleshy persons and children, and if neglected, it passes into eczema or an erythematous intertrigo.

Treatment.—The patient should be kept as cool as possible, and avoid undue perspiration. The food should be light and unstimulating, dispensing with meats and condiments for a few days; wine, spirits, and beer are to be avoided.

The ingestion of water, lemonade, Apollinaris water, Vichy water, together with refrigerant diuretics, as potassium citrate or acetate, a cool aprtment, and absolute rest will ordinarily insure speedy relief. Saline cathartics are invaluable.

Locally, sponging with alkaline solutions, dilute subacute of lead solution, fluid extract of grindelia (well-diluted), or a solution of witch hazel is beneficial. Cupric sulphate solution (gr. x to the ounce), carbolic acid (gr. xx) and glycerite of starch (℥ iii), and a dusting powder composed of lycopodium, starch, and zinc oxid may also be employed. The application of boric acid solution followed by boric acid powder is a valuable method of treatment.

SEBORRHEA.

Synonyms.—Acne sebacea; pityriasis; tinea furfuracea; dandruff.

Definition.—A functional disorder of the sebaceous glands of the skin, characterized by an excessive or diminished and abnormal secretion of sebaceous matter, forming upon the skin either as an oily coating or in crusts and scales.

Varieties.—*Seborrhœa oleosa; seborrhœa sicca.*

Causes.—In newly born infants an increased secretion of sebaceous matter—the *vernix caseosa*—is a physiologic process.

The origin of the disease is not fully understood, anemia being a factor in many cases. Brunettes are more often affected than blondes, and women more frequently than men.

Pathology.—Seborrhea is a functional derangement of the sebaceous glands; if it be allowed to become very chronic, there occurs atrophy of the glands and follicles.

Symptoms.—The affection may occur upon any portion of the body its most frequent seat being, however, the scalp (*seborrhœa captitis* or *pityriasis capitis*), and next in frequency the face (*seborrhœa faciei*).

Seborrhœa oleosa appears as an oily, greasy coating upon the skin, without hyperemia, and not attended with itching. The secretion is of an oily character, the quantity at times being so great as to collect in minute drops of a clear, yellowish fluid upon the surface. The most common seat for this variety is the face—*seborrhœa faciei*—and nose—*seborrhœa nasi.*

Seborrhœa sicca consists in the formation of dry, more or less greasy, masses of scales or crusts of a grayish, yellowish, or brownish-yellow color, having a strong tendency to adhere to the skin, and attended with decided itching. Occurring upon the scalp—*seborrhœa capitis*—it is a frequent source of premature baldness.

Diagnosis.—*Seborrhœa capitis* may be mistaken for dry eczema, but the former is always a *dry* disease, while in eczema moisture has occurred at some period of the affection. The scales in seborrhea are very abundant and pale; in eczema the scales are scanty and reddish, the parts irritated, infiltrated, and thickened.

Seborrhœa sicca and *psoriasis* have many points of resemblance, whether occurring on the scalp or on the body. In seborrhea the scales are minute or caked, grayish or yellowish in color, of an unctuous feel, and usually uniformly diffused. In psoriasis the scales are very dry, abundant, thick, white, irregularly dispersed, with intervening healthy skin, and the *surface beneath the scales is always reddish and inflamed.* The clinical histories of each are entirely different.

Prognosis.—If properly treated, favorable, although the affection is obstinate to eradicate. Its tendency to produce premature loss of hair when occurring on the scalp should be borne in mind.

Treatment.—The condition of the general health should receive attention. The secretions should be regulated. Anemia, chlorosis, gastrointestinal disorders, and other general conditions should receive appropriate treatment. Iron, arsenic, ichthyol, and calcium sulphid, internally, are of especial value in this condition. The following formula of Erasmus Wilson is often of benefit:

℞.	Vini ferri	f℥ iss	45. Cc.
	Syr. simplicis		
	Liq. potassii arsenit. .	āā fʒ ij	āā 8. Cc.
	Aquæ destil.	f℥ ij	60. Cc.

M. S.—Teaspoonful three times a day, well-diluted.

Local treatment is of greatest importance. In seborrhea of the scalp the scales and crusts should first be removed by olive oil, cod-liver oil, or lard applied at night and the head covered with a flannel or other cap. A mixture of boroglycerin (ʒ ii) and rose water (℥ viii) applied on gauze is also of value in this connection. As soon as the crusts are well soaked, they should be removed by washing with soap and warm water, or equal parts of soap, glycerin, and water, or the following:

℞.	Tinct. sapo mollis	f℥ iv	120. Cc.
	Spts. vini rect.	f℥ ij	60. Cc.

Solve et filtra.
M. S.—Dilute and use as a soap-wash or shampoo.

After removing the crusts, the scalp should be washed with warm water and carefully dried. In most cases, an ointment such as the following, rubbed well into the roots of the hair, is very beneficial:

℞.	Sulph. precip.	ʒ i	4. Gm.
	Acid. salicyl.	gr. x	0.6 Gm.
	Petrolat.	℥ i	32. Gm.

M. S.—Part the hair and apply directly to the scalp every night.

Or—

℞.	Hydrarg. ammoniat.	gr. xx	1.3 Gm.
	Lanolin	ʒ ii	8. Gm.
	Petrolat.	ʒ vi	24. Gm.

M. S.—Apply locally as directed.

The boroglycerid mixture mentioned above or the following combination is useful for dandruff:

℞.	Ammonii hydrochlorat. ..	gr. x	0.65 Gm.
	Glycerini	f℥ j	30. Cc.
	Aquæ rosæ	f℥ v	155. Cc.

M. S.—Apply to head.

Or—

℞.	Acid. boric	ʒ i	4. Gm.
	Acohol. (50 percent). ..	℥ iv	120. Gm.

M. S.—Apply locally.

Or—

℞.	Acid. carbol.	ʒ ss-ʒ i	2.-4. Gm.
	Ol. amygdalæ	fʒ iv	15. Cc.
	Ol. limonis	fʒ i	4. Cc.
	Aq. destillat. . .q. s. ad	f℥ ii	60. Cc.

M. S.—Apply locally after washing (Van Harlingen).

Or—

℞.	Resorcin	ʒ ss-ʒ i	2.-4. Gm.
	Ung. aquæ rosæ	℥ i	32. Gm.

M. S.—Apply locally.

Or—

℞.	Resorcin	ʒ ss	2. Gm.
	Olei. ricini	♏ xv	1.0 Cc.
	Alcohol.	f℥ iii	90. Cc.

M. S.—Apply locally by means of a medicine dropper. (For brunettes only).

Or—

℞.	Tinct. cantharidis	fʒ iij	12. Cc.
	Tinct. capsici	fʒ iij	12. Cc.
	Ol. ricini	fʒ ij	8. Cc.
	Alcoholis	f℥ ij	60. Cc.
	Spt. rosmarini	f℥ j	30. Cc.

M. S.—Apply locally (Duhring).

Or—

℞.	Bismuthi subnitratis	ʒ j	4. Gm.
	Ung. Hydrargyri ammoniat.	ʒ ij	8. Gm.
	Ung. aquæ rosæ	ad℥ j	ad 32. Gm.

M.

Seborrhea elsewhere, is treated in a similar manner, modifying the applications according to the individual needs of the case.

DISORDERS OF SENSATION.

PRURITUS.

A functional disorder of the skin characterized essentially by itching without structural alterations or obvious cause. Itching due to other disturbances is termed symptomatic and its relief depends upon the removability of the underlying condition. Primary pruritus is difficult to permanently cure but relief may be afforded by the use of applications containing carbolic acid, menthol, thymol, chloral-camphor, liquor carbonis detergens, and similar antipruritics. Idiopathic pruritus is by no means common and a careful search will usually detect some underlying condition for the apparent primary itching, removal of which relieves the pruritus. Frequently, cases without obvious cause come under observation which require symptomatic treatment while a search is being made for the underlying condition. For such cases the following formulas are applicable:

℞.	Acid. carbol.	ʒ ii	8. Cc.
	Glycerin	ʒ ii	8. Cc.
	Aquæ	Ə i	480. Cc.

M. S.—Poison, apply locally as directed.

Or—

℞.	Acid. carbol.	ʒ i-iii	4.-12. Cc.
	Glycerin.	ʒ ii	8. Cc.
	Alcohol.	℥ i	30. Cc.
	Aquæ	q. s. ad Ə i	480. Cc.

M. S.—Poison, apply locally as directed (Stelwagon).

Or—

℞.	Liq. carbonis deterg.	ʒ ii	8. Cc.
	Aquæ	℥ viii	240. Cc.

M. S.—Apply locally.

Or—

℞.	Thymol	ʒ ii	8. Gm.
	Liq. potass.	ʒ i	4. Cc.
	Glycerin	ʒ iii	12. Cc.
	Aquæ	℥ viii	240. Cc.

M. S.—Apply locally (Crocker).

Or—

℞.	Resorcin	ʒ ss	2. Gm.
	Glycerin	ʒ i	4. Cc.
	Liq. calcis	f℥ iv	120. Cc.

M. S.—Apply locally.

Generalized itching nearly always suggests infection by some of the animal parasites, especially scabies. When the scratch-marks are localized for the most part to the flexor surfaces it is a good plan to advise the following:

℞.	Sulph. precip.		
	Betanaphthol	āā ʒ ss	āā 2. Gm.
	Petrolat.	℥ i	32. Gm.

M. S.—Apply to all parts of the body, excepting the head and face, for four nights; then follow with a bath. Should the itching then continue, do not repeat the treatment at once, but instead use a carbolic acid lotion for a week or ten days and then return to the first treatment if necessary.

Menthol is frequently of value in relieving itching, especially when incorporated in an ointment or paste.

℞.	Menthol	gr. viii	.48 Gm.
	Pulv. amyli		
	Pulv. zinci oxidi	āā ʒ ii	8. Gm.
	Petrolat.	ʒ iv	16. Gm.

M. S.—Apply locally.

Brocq advises the following formulas for this condition:

℞.	Resorcin	gr. iv	0.25 Gm.
	Hydrarg. chlorid. mit.	gr. xii	0.75 Gm.
	Zinci oxidi	gr. xxx	2.00 Gm.
	Petrolat.	ʒ v	20.00 Gm.

M.—Ft. ung.

S.—Apply locally.

℞.	Menthol	gr. iii	0.2 Gm.
	Acidi carbolici	gr. iv	0.25 Gm.
	Acidi salicyli	ʒ ss	2.00 Gm.
	Zinci oxidi	ʒ iss	6.00 Gm.
	Liq. petrolat.	℥ i	32.00 Gm.

M.—Ft. ung.

S.—Apply locally.

Pruritus ani is perhaps the most distressing form of this disease and for its permanent relief, most careful attention must be given to the most minute details of the case. The following formulas are recommended for relief of the itching:

℞. Hydrarg. ammoniat. ....gr. xx 1.2 Gm.
Adeps benzoinat. ....℥ i 32. Gm.
M. S.—Apply locally (Crocker).

℞. Menthol,
Chloral,
Camphor ....āā gr. v .3 Gm.
Petrolat. ....℥ ss 16. Gm.
M. S.—Apply locally.

℞. Cocain hydrochlorat. ....gr. xv 1. Gm.
Bismuth subnitrat. ......gr. xxx 2. Gm.
Lanolin ....ʒ v 20. Gm.
M. S.—Apply locally.

When complicated with hemorrhoids, the following is often of value:

℞. Ext. hamamelis fld. .....ʒ viii 30. Cc.
Ext. hydrastis,
Ergotin ....āā ʒ xv 60. Cc.
Tinct. benzoin ....ʒ xv 60. Cc.
Olei olivæ ....ʒ viii 30. Cc.
Acid. carbol. ....gr. xxiii 1.5 Cc.
M. S.—For external use.

Malcolm Morris speaks favorably of the following combinations in the treatment of pruritus ani:

℞. Acidi carbolici ....♏xx 1.2 Cc.
Cocain. hydrochlorat. ...gr. x .6 Gm.
Vaselin ....℥ i 32. Gm.
M.—Ft. ung.
S.—Apply locally.

℞. Ung. picis liquidæ ......ʒ i 4. Gm.
Bismuthi subnitratis ....gr. xx 1.2 Gm.
Adipis ....q. s. ad ℥ i 32. Gm.
M.—Ft. ung.
S.—Apply locally.

℞.	Acid. carbol.	ʒ ss	2.	Gm.
	Hyd. perchlor.	gr. ii	.12	Gm.
	Ol. olivæ	ʒ ii	8.	Gm.
	Ung. zinci oxidi benzoat. q. s. ad	℥ i	32.	Gm.

M. S.—Apply locally.

Laplace advises the following formula for certain cases of anal itching:

℞.	Hydrarg. chlorid. corrisiv.	gr. ii	.12	Gm.
	Acid. hydrochlor.	♏ x	.12	Cc.
	Aquæ	f℥ viii	240.0	Cc.

M. S.—Apply locally.

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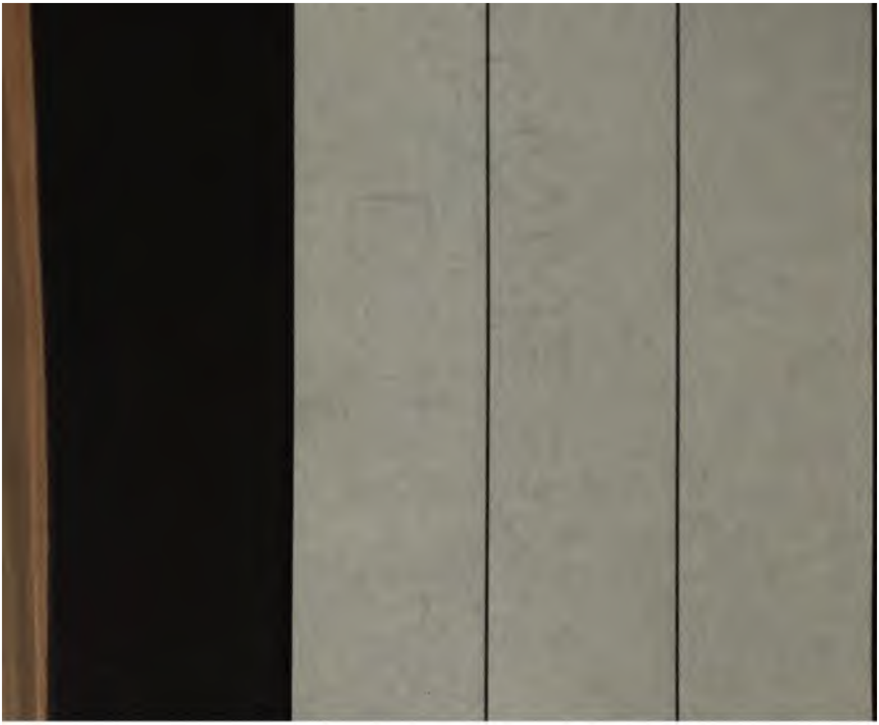

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